CHEST EXAMINATION

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THE CORRELATION OF PHYSICAL AND X-RAY FINDINGS IN DISEASES OF THE LUNG

By

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FOREWORD

WHILE still a medical student I felt strongly that the next generation would condemn the practice of herding cases of proved pulmonary tuberculosis in out-patient departments. There they would sit in rows among the other patients, coughing over them, ultimately to be rewarded by a bottle of cod liver oil and some linctus. But we did have the opportunity of learning our physical signs from these unfortunates. To-day I can affirm from my experience as an examiner at various universities that the weakest part of the candidates' equipment is their recognition and interpretation of physical signs in the lungs. To some extent this may be due to the segregation of tuberculous cases into departments where students do not freely enter, but still more to their tendency to short-circuit their examination by flying to the X-ray film, using it as a substitute instead of a confirmation of ordinary clinical methods. The teachers, however, are not entirely free from blame, particularly in the matter of multiplying labels for the various sounds heard through the stethoscope; the degree of accuracy thereby implied is largely illusory, and merely confuses the student. It is one of the many merits of Wing Commander Trail's book that he follows the tradition of my own teachers, Dr. Gee and Dr. Samuel West, by using as few terms as possible, and correlating each with its pathological significance.

This plan is characteristic of the whole work; anatomy, physiology, pathology, symptoms, physical signs and X-ray findings are closely correlated, enabling the student or practitioner to build up a mental concept of exactly what is happening inside the chest. Many will be interested to learn how greatly modern X-ray technique has advanced interpretation in skilled hands. Even more will be surprised to find what helpful deductions from physiological principles they habitually ignore. And all will be relieved to find how a grasp of the first principles involved will enable them to arrive at a correct opinion without imposing burdensome details on their memory.

As a former teacher I know how valuable a contribution this book makes to the understanding of vital problems, while the freshness of its approach will be patent to all.

W. LANGDON-BROWN.

PREFACE TO THE SECOND EDITION

THE Second Edition has been slightly enlarged, particularly by the addition of illustrative X-rays, set in the text so that they are easy of reference. The order of Contents has been changed, and notes added on such points as the interpretation of breath sounds and the "normal abnormalities" of the bony thorax.

These alterations are due to the helpful criticism of notices in the Medical Journals, and in particular to the kindness of many readers who wrote personally to the author. Among the latter I wish to thank especially Dr. J. D. Grove-White, Dr. R. L. Midgley, and Dr. H. Courtney Gage, who sent their detailed observations as a general practitioner, a chest physician, and a radiologist respectively.

R. R. TRAIL.

PREFACE TO THE FIRST EDITION

THIS book is founded on courses of lectures which it has been the writer's good fortune to give to students and post-graduates. They owe their present form in great part to attempts made to answer the difficulties of those students who showed an interest in an approach to examination of the chest founded on a knowledge of applied anatomy, physiology and pathology, without which it is naturally useless to attempt a reasonable interpretation and correlation of physical and X-ray findings.

Correlation is not so difficult to acquire as many students seem to fear. Much confusion has arisen of late years because of the big advances made by chest physician specialists on the one hand and chest radiology specialists on the other. More recently, however, there has been a tendency to combine these branches in the "chest physician cum radiologist," or the chest physician who likes to read his own X-rays. This is all to the good of the modern student; it tends to bring us back to fundamentals. We find it increasingly easy to go beyond the objective reading of films. We can connect physical signs with definite

abnormalities of shadow, and combine both with the changes in normal anatomy and physiology so ably expressed nowadays by the specialised lung pathologist, who is, we must remember, but the first offshoot of that type of learned physician who laid his firm foundations for practice in his earlier post-mortem room researches, when X-rays did not exist.

The first Section of the book is, therefore, devoted to reminders on those salient points of normal anatomy and physiology which explain the abnormalities of the commoner chest diseases of general practice. Like other points of equal importance, they are repeated by reference to other sections.

In the second Section on Applied Pathology we consider the main gross and microscopical changes induced by these diseases, and, in noting the physical signs and the alterations from the normal postero-anterior film that accompany them in their various stages, an attempt is made to correlate all three aspects. A certain amount of detailed description is necessary, but this is confined as much as possible to fundamentals, even if by this statement we appear to be begging the question.

There has been of late years much discussion on the reading of abnormal films, and of necessity so much individual variation in reports, that an attempt has been made in some quarters to confine them to objective wording. As already indicated, the writer feels that we can go further; that we should attempt to arrive at criteria for interpretation, using the work of the physician, the pathologist and the radiologist as a combined whole. We shall all agree that changes of microscopical detail cannot be expected to reflect themselves in stethoscopic signs and on the usual postero-anterior film; we know that a lobule on the lung surface is no more than about one-quarter of an inch in its longest diameter. Nevertheless, it is felt that a knowledge of pathogenesis is fundamental for the student who would correlate his physical and X-ray findings, even if these are demonstrable only when comparatively gross areas of lung are involved. Thus we know that in phthisis stethoscopic signs are late, but we can find a reason why they are late and so why we should look for other and as important physical signs, such as lack of movement and note, that will precede these stethoscopic signs, and so warn us of the presence of a specific pathological change. It may be argued that the prime correlations here detailed are built on *a priori* reasoning; even if this be justifiable criticism it will be

admitted that they can give some elementals that may combine the still too-well defined compartments of the pathologist, the radiologist and the physician. Even a mere workable explanation would be better than no basis at all.

Section III sums up the physical findings in the diseases discussed in Section II, and gives a scheme of interpretation of stethoscopic findings that may act as a basis for diagnosis without X-ray findings. No attempt is made to deal with all normal and abnormal stethoscopic signs, but only with such adventitious sounds as can be correlated with underlying pathogenesis. It is taken for granted that the student has listened to large numbers of normal chests and a sufficient number of abnormals to understand the principles underlying the interpretation of breath sounds and voice sounds; *e.g.*, the difference between the prolonged expiration of emphysema and the high pitch, faint or loud to the ear, that connotes bronchial breathing.

It will be seen that all these sections are inter-connected. They are in essence the parts of an interesting jig-saw puzzle. True they place personal values on the individual pieces used in the game, but they are the result of the writer's conscious attempt over several years to increase the value of each piece by using experience to alter its shape so that it fits more easily with its fellow. Each new problem in living pathogenesis continues to be instructive, while it whets the appetite for still further problems.

The ultimate aim of this handbook is to bring added help to the student undergraduate and post-graduate, in the assessment of the living and changing pathogenesis of his individual patient; only by so doing can we arrive at a reasoned basis for that wider problem of individual treatment, which must be the object, and is always the only true reward, of the happy physician.

My thanks are due to Sir Walter Langdon-Brown for his kindness in writing a Foreword, to F/Lt. J. A. Kennedy for his assistance in reading proofs and in preparing the index, and to Mr. J. Rivers of Messrs. J. & A. Churchill Ltd. for his valuable help in these days of difficulty with paper and print.

R. R. TRAIL.

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CHEST EXAMINATION

SECTION I. APPLIED ANATOMY

CHAPTER I

THE BRONCHIAL TREE

The trachea lies behind the great vessels, embedded in the elastic areolar tissue which ensheathes all the structures lying in the mediastinum. It divides at about the level of the fifth dorsal vertebra into its two main branches, the right and left bronchi. To this point it can be seen on the normal postero-anterior film of the chest as a clear area, bearing slightly to the right of the mid-line of the thorax.

The point of division is important in all conditions which cause displacement of the mediastinum, in that it lies not far below the third dorsal vertebra, opposite which is the weakest part of the mediastinum. On this point the mediastinum swings laterally, as on a hinge. Any pull on the lung structures connected with one main bronchus is easily transmitted along the lower part of the trachea to this weak point. We shall see later that the trachea responds more than any other mediastinal structure to lung changes, and that therefore if we find evidence of its disturbance we can reason backwards to find the cause in such lung changes. Fortunately its movement is quickly reflected on the sternomastoid muscle so that we have an easily demonstrable sign of its displacement.

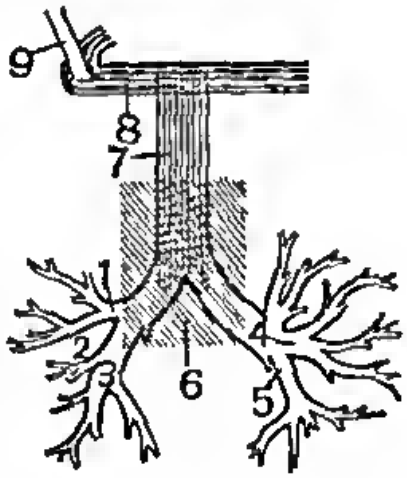


FIG. 1. The Mechanism of the Sternomastoid Sign.
1. The right upper lobe bronchus.
2. The right middle lobe bronchus.
3. The right lower lobe bronchus.
4. The left upper lobe bronchus.
5. The left lower lobe bronchus.
6. The areolar tissue of the mediastinum.
7. The pretracheal fascia.
8. The fascia of the neck.
9. The sternomastoid muscle.

The anatomical relations of the muscle explain this reaction (see Fig. 1). On the anterior surface of the trachea lies a band of tissue called the pretracheal fascia, connected below with the elastic areolar tissue of the mediastinum, and above

1

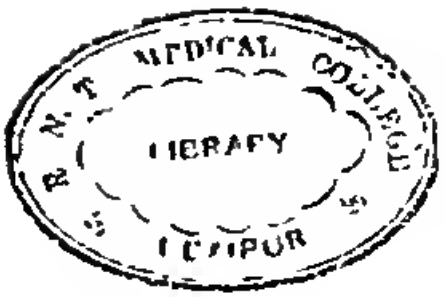
with the deep fascia of the neck. The latter fascia, which meets from both sides in the middle line, divides as it goes backward, to enclose the sterno-mastoid muscle, and is thus in close contact with its tendinous part which has its origin at the anterior superior border of the manubrium sterni. We see therefore how it is that tracheal displacement is reflected to an increased tension of the sternomastoid muscle in this tendinous part, on the same side as that to which the mediastinum is pulled or pushed, by traction on one side or pressure from the other.

As we proceed with our studies we shall find that a pull on the trachea is effected by all processes which interfere with the elastic tissue of the lung. This elastic tissue is continuous throughout the bronchial tree, and carried from its smallest division to the elastic surround of the air cells; indeed, with the finest, ultimate arterioles, it forms the actual alveolar wall.

All branches of the bronchial tree are also intimately surrounded by a binding of connective tissue, which acts as a supporting structure like a scaffolding. It is resilient, and moves in response to movements of the bronchi, as they alter in length and diameter by inspiration and expiration; but it is not an integral part of the bronchus like the elastic tissue, and so its pathological changes have not the same opportunity to reflect themselves on the mediastinum. Its changes appear to act only secondarily. Its commonest pathological change is in the deposition of fibroblasts, which produce péribronchial fibrosis. Increase in depth of tissue leads only to ultimate shrinkage and loss of resilience; what seems to happen is that the fibrosis obliterates lobules it was meant to support, and causes such a drag on others in its neighbourhood that it destroys their elastic tissue after distending them.

Direct and indirect destruction of elastic tissue occurs quite early in adult pulmonary tuberculosis. Material from diseased lobules enters, and then blocks, the supplying terminal bronchiole. As air cannot now reach the alveoli, empty ones collapse, and full ones organise, and both lose their elasticity. The trachea is thus pulled to the diseased side, and we find a sharp inner border to the sternomastoid muscle on the same side.

The effect of the mediastinal hinge on tracheal displacement is well shown in disease confined to the upper lobes. It is not unusual to find that the part above the third dorsal vertebra is dragged into a definite bow by localised infraclavicular tuberculosis. In the same way, a kink in this region is seen in several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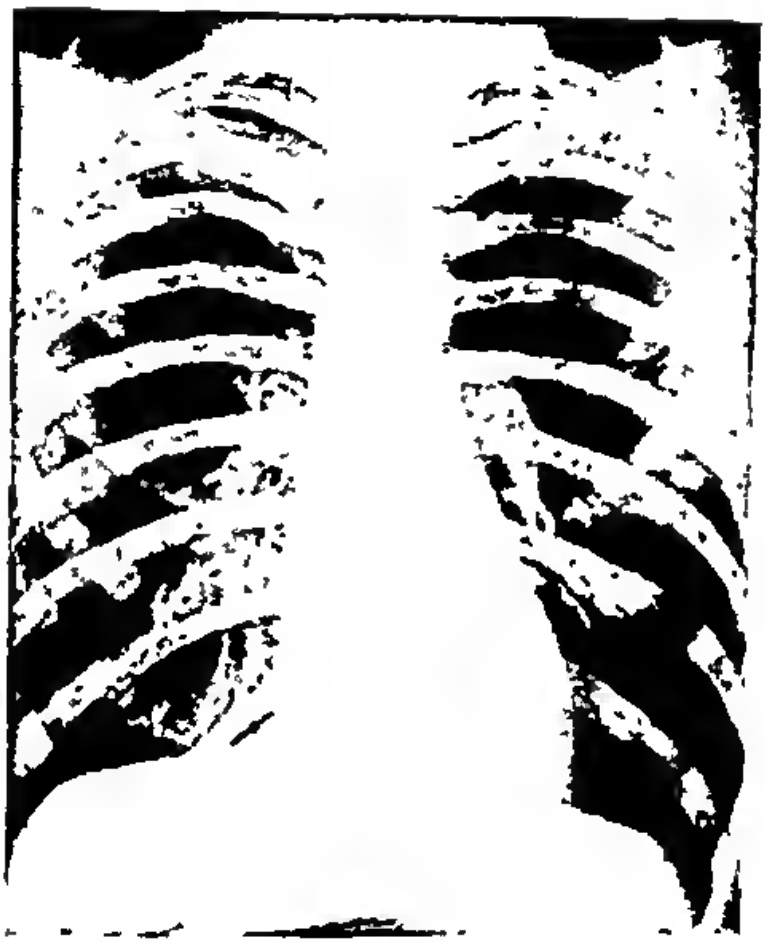

FIG. 3. Normal postero-anterior film.

[To face p. 2

cases of substernal thyroid, and in adenoma of one lobe of the gland, which pushes the trachea out of its central position.

Along with other mediastinal structures, the trachea is displaced in the common abnormality of dorsal scoliosis, which is usually convex *to the right. This is really a torsion of the thoracic* cage, pushing the right lower half forwards and outwards, and pulling the left lower half inwards and backwards, as viewed anteriorly. When we look at the patient from the front we see his right lower ribs forced apart to produce bulging, while the left lower half appears to be fallen in; exactly the opposite is found on looking at the back; the right lower zone is flatter, and the left lower zone more prominent than the normal. In other words, the volume of the chest cavity as a whole is not reduced. The effect of this torsion on the shadow of the heart and of the main vessels will be discussed in the chapters on the mediastinum in health and disease, and on the normal postero-anterior film.

It is interesting to note that the effect of scoliosis on rib-spaces is no longer seen when pulmonary tuberculosis supervenes. Compensatory emphysema seems to undo it.

For all practical purposes the student can consider the bronchi as dividing by dichotomy down to their smallest branches: that is, each bronchial division divides into two smaller branches, and this process continues until we reach the bronchioles. We cannot follow these divisions on the chest film, but we can see the divisions of the vascular supply of the pulmonary artery, and these are so similar, and so closely approximated to the bronchial divisions, that they form a practical guide. They can be traced on a good postero-anterior film throughout three-quarters of the lung fields from their origins in the hilar regions (Figs 2 and 3).

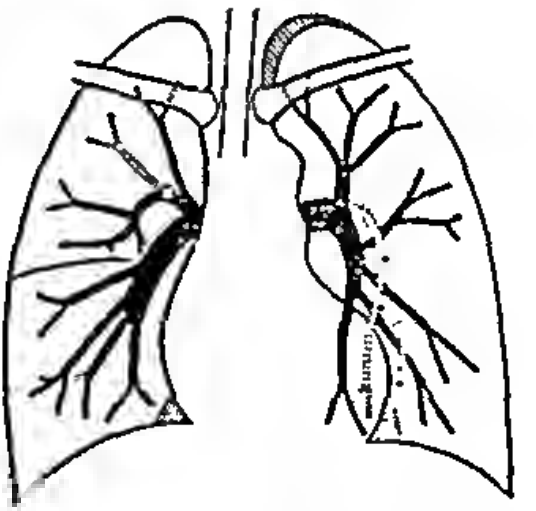

FIG. 2. The Blood Supply. The posterior branch of the right upper lobe blood supply, and the blood supply to the lingula of the left upper lobe are shown in dotted lines.

There is one point of difference that is important when reading abnormal films: blood vessels continue below the shadow

of the diaphragm; bronchial shadows do not, unless they are enlarged by disease.

The diagram of the blood supply shows the branches of the pulmonary arteries as they appear on a good postero-anterior film. The right pulmonary artery makes a T-shaped shadow at the hilum. It gives off three branches to the upper lobe, one to the middle lobe, and three to the lower lobe. The left artery gives an elbow-shaped shadow at the left hilum, about half an inch higher than the right one on a 15 × 12 inch film. It gives five branches to the upper lobe, and four to the lower lobe. It will be noted that much of the left lower lobe supply is hidden by the heart shadow, and that the lower part of the upper lobe is supplied by two branches, convex outwards, that come well down towards the

Fig 4.

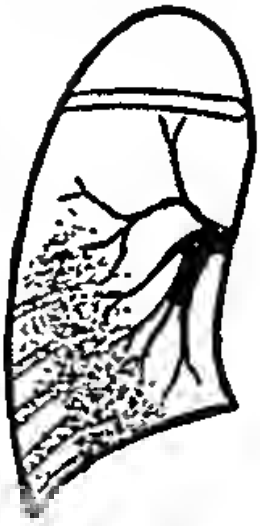

Fig. 5.

Fig. 6.

left cardiac border, supplying the thin triangular part of the lobe that lies in front of the lower lobe.

If any of these normal markings are altered in distribution, or have disappeared, we can conclude which part of the bronchial tree has been interfered with by disease, and so get a very helpful aid to diagnosis.

Thus on the film of right lower lobe pneumonia we see no shadows of the blood supply to the diseased lobe. If we can follow the markings of the rest of the blood supply to the lung we shall find them quite normal in their distribution (see Fig. 4).

If the disease is right pleural effusion we can find the shadows of congested blood vessels in the compressed lung, internal to the shadow cast by the fluid. There is no obliteration of blood supply (see Fig. 5).

On the film of pleural effusion complicating lower lobe pneumonia we shall make out the shadow of the fluid towards the periphery ; internal to it we see no markings of blood supply in the diseased lobe. The rest of the blood supply is normal in distribution (see Fig. 6).

If we are dealing with a case of collapse of the right lower lobe we shall note two effects on the shadows of the blood supply. First, there are no markings in the area of collapse, which is more or less opaque, and appears to be continuous with the heart shadow. Second, as the lower lobe has collapsed and shrunk, the space it has vacated is filled by the upper and mid lobes, which

Fig. 7.

Fig. 8. Collapse of the right upper lobe.

enlarge by emphysema, so that their blood supply is splayed out as against its normal distribution (see Fig. 7). Fig. 8 shows the effects of collapse of the right upper lobe. There is no sign of the blood supply to the collapsed area ; that to the middle and lower lobes is spread laterally as against the normal distribution.

Now and again we come across subjects in whom the normal "dichotomous" division of the bronchi has gone wrong. Development has ceased at one point of bifurcation and instead of a branch we have an air-containing space, blown out by air from the bronchus and kept open by surrounding lung structures. It may be small or large, and is lined by the normal elements of the bronchus. It is known as a "congenital cyst of the bronchus." It can be found in forms which maintain their connection with the original bronchus, but many are closed off, from the first, or

later in life. Some contain a serous sterile fluid, but most are empty. The walls are thin but well enough defined, and the bigger ones may give demonstrable pressure on surrounding structures. When the connection with the original bronchus is maintained through a valvular-like flap these cysts may have a clinical significance, but the usual form has no effect on the subject. On the film these appear as single or multiple circles, sometimes superimposed on each other like heaps of thin curtain rings (Figs. 9 and 10).

Fig. 9. Showing pneumonia of the pectoral and axillary branches of the right upper lobe, and cystic disease of the right lower zone.

We must remind ourselves of certain of the principal divisions of the bronchi, because they explain to us localised lung pathology. Each of these divisions supplies a fairly large cone-shaped area of lung tissue, the apex of the area being at the entrance of the branch. This is why disease of any one such area, which lies laterally in the lung, appears on a postero-anterior film of the chest as a triangle, and why from the position of such a triangle we can deduce the actual bronchial division involved in, *e.g.*, collapse of certain lobules by bronchial occlusion. Four such branches are of particular interest to the student, all in the right lung, three being connected with the upper lobe bronchus, and one with the lower lobe bronchus.

The right upper lobe, or epiarterial bronchus, has three main divisions: the apical, the axillary and the pectoral. The apical branch goes upwards, outwards and a little backwards. It supplies that part of the upper zone of the lung which is weak in supporting structure, and so is a common site for emphysema and for the first signs of lobar collapse from bronchial blockage. Its air-containing cells soon fall in when they are not kept fully supplied by residual air, which is their principal method of maintaining their position. They may get stretched and become emphysematous by their lack of support, and when they rupture by such stretching may cause a tearing of the visceral pleura to produce a spontaneous pneumothorax. This type of pneumothorax is known as "simple," to distinguish it from pneumothorax of tuberculous origin (see Fig. 11).

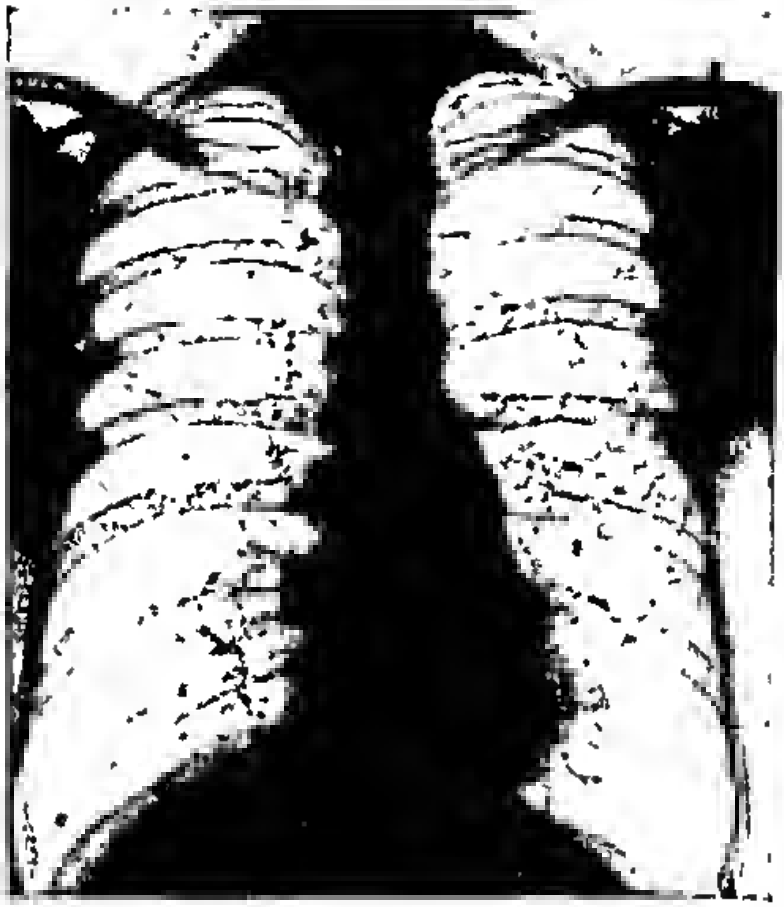

FIG. 10. Congenital cystic disease complicated by collapse and bronchiectasis in the left lower lobe.

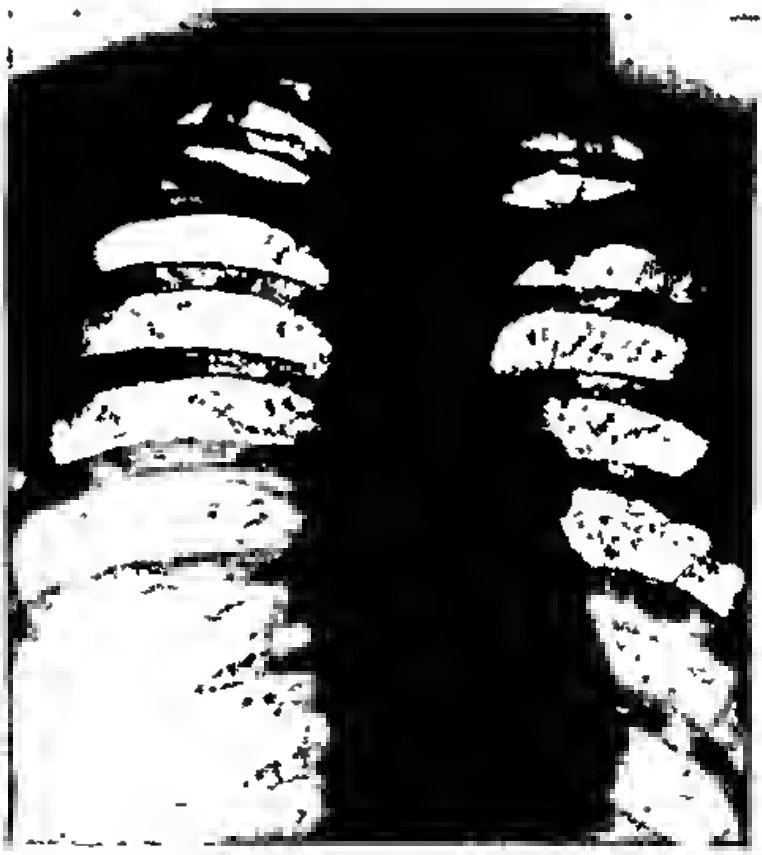

FIG. 11. Simple spontaneous pneumothorax, with emphysematous bullæ on the upper border of the collapsed lung.

Fig. 13. Apical tuberculosis of the left lung.

The apical branch is not often affected by pneumonia, but is especially important in that it gives off a dorsal branch supplying a sector of the parenchyma commonly affected in adult tuberculosis. When this occurs we see in the supraclavicular area of the lung field small rounded deposits, more or less defined in outline and dense in the centre. Reference will be made later to the difficulties in interpretation of shadows in this area. In the meantime it is sufficient to say that *it is a fairly safe rule not* to read evidence of apical tuberculosis unless one can see quite definite areas of loss of translucency, or at least five rounded dots lying under, internal or external to the shadow of the anterior end of the first rib, above the collar-bone (see Figs. 12 and 13).

The axillary branch comes off the main trunk close to the pectoral branch, towards the centre of *the upper mid-zone of the* right lung field, and is directed outwards to supply the outer area of the lobe below the clavicular zone. When this branch is involved in such a disease as pneumonia we have therefore on the film a triangular area of loss of translucency, which has its apex at the entrance of the branch, and its base along the axilla (see Fig. 9, p. 6, and Fig. 52, p. 78). Like the apical branch, it gives off a dorsal bronchus important in adult tuberculosis.

FIG. 12. Common sites of tuberculosis in broncho-*pulmonary segments* supplied by dorsally inclined bronchi.

Later, in the section on applied pathology, we shall discuss the differential diagnosis of the film shadows of "partial" pneumonia involving the axillary bronchus, and of adult pulmonary tuberculosis at the outer base of the right upper lobe.

The pectoral branch bends a little outwards and then comes forward in a semicircular fashion; the arching of the accompanying branch of the *pulmonary artery sometimes shows up prominently* in a completely normal film as if it were half a cavity wall, the other half being formed by the mediastinal shadow. If the lung sector of this bronchus is involved in pneumonia there is a triangular loss of translucency rather like a mirror image of the axillary sector; this time the base is against the mediastinal shadow and the apex towards the centre of the upper-mid zone of the lung.

In some cases the shadow is first noted when the apex of the triangle is rounded off; this may cause considerable difficulty in diagnosis as against tumour or cyst. Serial films usually clinch the diagnosis, by the comparatively rapid changes that occur in the "partial" pneumonia as the disease resolves (see Fig. 9. p. 6, and Fig. 14).

The principal branch to remember from the right lower lobe bronchus is the posterior horizontal. It does exactly what its name describes. It comes off the main stem of the lower lobe bronchus posteriorly, practically opposite the origin of the middle lobe bronchus, and therefore near the centre of the hilar shadow on the film. It goes almost horizontally backwards. If, therefore, it is involved in disease, the abnormal shadow is close to the hilum. This is why so-called central pneumonia, commoner in children than in adults, appears to be part of the mediastinum. As it hooks backwards over the apex of the lower lobe this branch is a common site for blockage. Its lung segment is often involved in adult phthisis. Septic material is easily caught in it to give the foundation for an abscess, while the accompanying lymphatics round its blood supply are equally easily occluded by phagocytes containing tubercle bacilli.

FIG. 15.

Its occlusion is a common cause of a characteristic film of adult tuberculosis. When a tuberculoma has formed in this area, close inspection of the postero-anterior film will often reveal a line running downwards and outwards from the hilum, and forming the outer boundary of a triangular loss of translucency. The lingula of the lower lobe has shrunk downwards and inwards on to the main interlobar fissure. There is collapse of the apex of the lower lobe (see Figs. 15 and 16).

From the foregoing outline it will be seen that broncho-pulmonary segments supplied by divisions of the bronchi that go backwards are sites of election for adult tubercle; we must know particularly the dorsal branches of the apical, axillary and pectoral divisions of the epiarterial bronchus, and the posterior horizontal branch of the right lower lobe bronchus. They are similarly the earliest sites for evidence of blockage of lymphatics in diseases of dust inhalation, such as silicosis. The broncho-

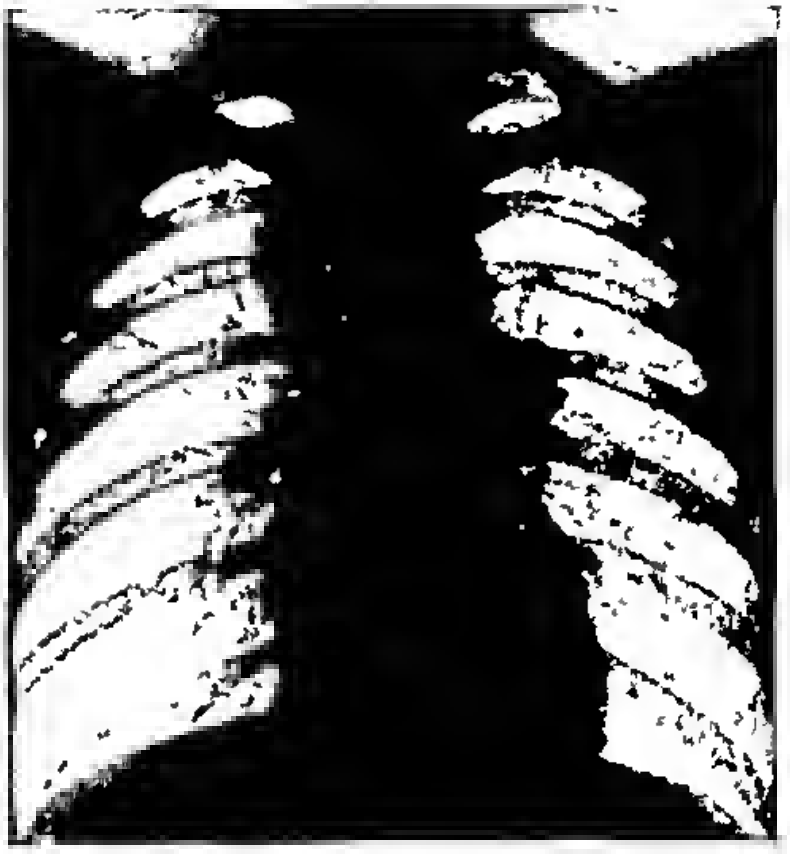

FIG. 14. Pneumonia of the pectoral branch of right upper lobe bronchus

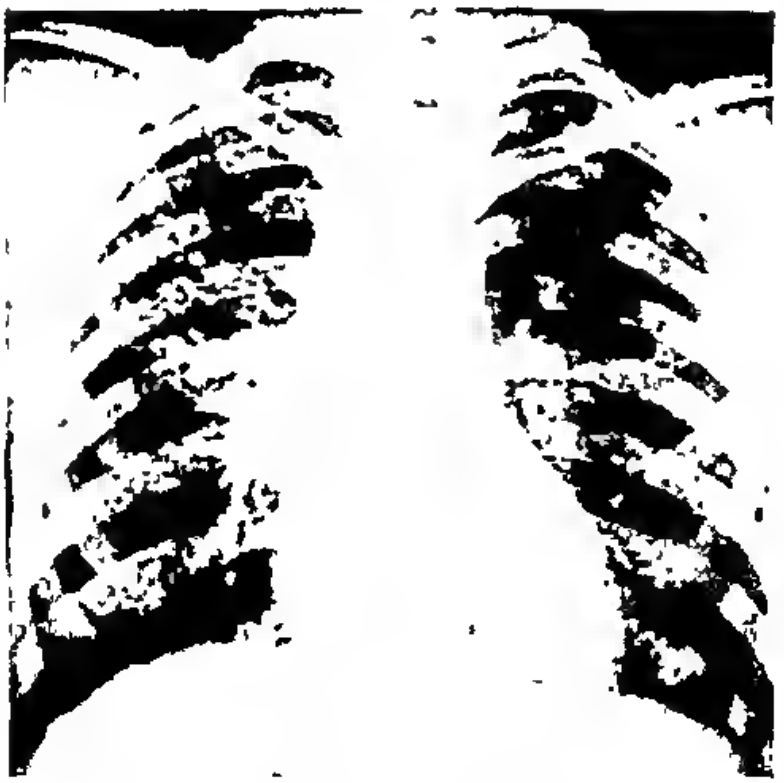

FIG. 16. Pulmonary tuberculosis. The collapsing apex of right lower lobe as a tongue-shaped shadow in third interspace.

[*To face*]

FIG. 17. Assmann's focus in left lower lobe.

pulmonary areas supplied by these branches are, like all such areas, conical in shape, but on the postero-anterior film are photographed along a line drawn through their narrowest point at the entry of the bronchiole. It follows, therefore, that disease in them throws a rounded or oval shadow, because the bronchus and also, therefore, the segment of lung it supplies, lie antero-posteriorly in the chest. An antero-posterior film (*i.e.*, one with the patient's back to the screen) will show how the diseased area goes backwards (see Fig. 12, p. 7, and Fig. 17).

It is important to note that the right middle lobe bronchus comes off the right main bronchus at an angle of almost 90 degrees, and that its branches go out almost horizontally towards the periphery of the lung. This makes the sectors of parenchyma supplied by its bronchioles appear as small triangles with their apices inwards and their bases outwards when they are affected by disease (see Figs. 57 and 58, p. 80). It also makes them, as their main stem, difficult to drain and easy of blockage, and we shall find in this the explanation for the comparative frequency of complications, in this lobe, of diseases which in other lobes of the lung give no such cause for worry to the physician.

We have already noted that all branches of the bronchi carry throughout the lung elastic tissue continuous with that of the trachea. By this elastic tissue they are able to return to their normal position of rest after they have been elongated and opened up in the act of inspiration. It follows, too, that by such distribution the elastic tissue is anchored to the hilum, so that the lung is always willing, the more it is pulled outwards, to return to this point of anchorage.

Similarly, all branches carry the tracheal structures of cartilage and muscle, and have round them sheaths of connective tissue, supporting them, but moving with them in inspiration. These sheaths carry the blood vessels from the bronchial artery, and are closely related to the accompanying circulation from the pulmonary artery. Through them run also the lymph channels. They send out strands which support the lung parenchyma. We shall consider the connective tissue in more detail when dealing with the structure and functions of the alveoli.

CHAPTER II

THE ALVEOLI: THEIR STRUCTURE AND FUNCTIONS

While the bronchi continue to divide they retain their component cartilage, muscle and elastic fibres into the final subdivisions called the terminal bronchioles. There is much argument among research workers on the details of what exactly happens thereafter until we reach the air cells or alveoli, but what matters to the student is that, while all the bronchial elements remain up to the terminal bronchiole, the cartilage disappears after this subdivision, but strong muscle and elastic fibres continue into the final cylindrical channel, the bronchiolus respiratorius. This bronchiole with its communicating air-cells forms that minute section of lung called the acinus (see Fig. 18).

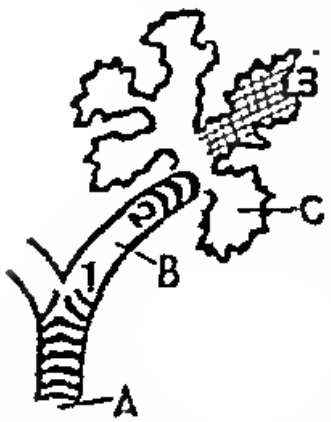

Fig. 18. The Acinus.
1. Termination of cartilage.
2. Termination of muscle fibres.
3. Termination of elastic fibres.
A. Terminal bronchiole.
B. Bronchiolus respiratorius.
C. The acinus.

The Acinus

The acinus is so important in the understanding of many diseases of the chest, and is the basis of explanation of so many physical and X-ray findings, that we must pay some particular attention to its details of construction, and its relations both anatomical and physiological.

Its bunches of air cells, or alveoli, contain the "residual air" of the lung, that is, the air that remains after a maximal expiratory effort has been made to expel what is called "supplemental air." In other words, this residual air can be expelled only by collapse of the cells. It is in contact with cubical epithelium, which has replaced the columnar ciliated epithelium of the bronchi, and through it is brought into close contact with the finest capillaries of the blood supply for gaseous interchange.

Residual air is at atmospheric pressure, being the same as that of inspired air, and therefore the same as that continually pressing on the outside of the thoracic cage. In other words, it is

one offset to the attempt of outside air to push in the bony thorax. The same ultimate effect would be got by any other occupying material of the alveoli, *e.g.*, pneumonic exudate, as long as the pressure is the same. This we shall see is an important point in the differential diagnosis of chest disease.

The muscle fibres continue along the bronchiolus respiratorius and get steadily stronger until we reach the last passage that can be said to be a lumen, where, we must note, they finish by becoming circular only, instead of longitudinal and circular. This anatomy explains many fundamentals of normal physiological lung movement and of the changes instituted by disease processes. By the act of inspiration there is a lengthening of the muscle fibres throughout the whole bronchial tree as far as the acinus; thus there is stretching and opening up of every air-containing lumen. The muscle is "smooth muscle," and so maintains movements along the bronchi even when the lung is at rest, thus keeping up the flow of air to the alveoli, with an action similar to that of the bagpipe.

Destruction of muscle in the bronchial wall is therefore of serious consequence to the acini; their residual air is not maintained and they are apt to collapse. Muscle spasm in such a disease as asthma has also a serious effect; the steady spasm induced by the disease, even during lung rest, is accentuated by the movements of inspiration, and is communicated to the circular fibres, which close in vice-like fashion over the entrance to the acinus. The patient has therefore increasing distress by his difficulty in maintaining sufficient residual air. Such an addition to his already established emphysema explains the extreme cyanosis often noted in the "bronchial asthma" of the chronic bronchitic. This point will be better understood when we consider the effects of emphysema.

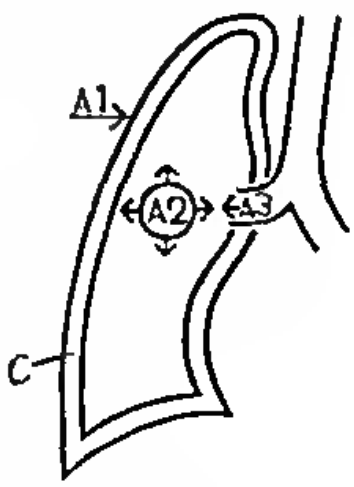

FIG. 19.

A_1. Air pressing on the chest wall.
A_2. Air in alveolus.
A_3. Inspired air in main bronchus.
C. Inter pleural space.

Elastic tissue continues all the way to the alveoli, finishing round each as if it were a net-bag round a tennis ball. Continuous from the main bronchial division in the mediastinum, which may be considered as its point of anchorage,

it is kept on constant stretch in every direction throughout the lung fields by the air in the alveoli (see Fig. 19, p. 11). What this comes to is that the residual air in each alveolus is like a small distensible bladder, which has two physiological effects. First it keeps up tension on the elastic tissue, so that the pull on the anchorage is constant, and therefore the lung is always attempting to retract towards the hilum. Second it has a marked effect on the strength of the outward pull of the pressure in the pleural cavity, which is below that of the atmosphere, and is therefore called the "sub-atmospheric" or "negative intrapleural pressure." The maintenance of residual air at a definite level is thus a necessity for proper physiological working of the lung, both at rest and during the act of inspiration. If it is cut off by blockage of its supplying channel, or destroyed by the breaking down of its alveolar confining walls, we shall find definite changes in the position of the lung and the mediastinum.

A few examples will help us to understand the ultimate effects of certain disease processes, by noting the physiological effect of their pathological changes on lung elasticity.

(*a*) Destruction of elastic tissue along the bronchial wall occurs in bronchiectasis and tuberculosis. As some of the tension is thus removed from the point of anchorage of the elastic tissue at the main bronchial division in the mediastinum, the lung medial to such a break in continuity tends to contract towards the hilum.

(*b*) Destruction of muscle in the bronchial wall is seen in tuberculosis and bronchiectasis; air is not maintained in those lobules dependent for supply on the affected branch, because the bagpipe action of the smooth muscle has been lost, and the end-result is the same as in (*a*).

(*c*) Cutting off residual air, which maintains the stretch on the elastic surround of the alveoli, happens if anything blocks the supplying lumen—pus, blood or a foreign body. This happens in adult phthisis. Tuberculous material from diseased alveoli enters, and later blocks the bronchiole supplying them. Empty air cells then collapse; those that still contain material organise, and all lose their outward pull on the mediastinum.

The acini are bound together into lobules by connective tissue septa. This connective tissue, which runs throughout the lung as a sheath accompanying the bronchial distribution, acts as the supporting structure. It binds bundles of air-cells together into lobules, and acts as a barrier which tries to confine disease to

FIG. 20. Early bronchiectasis. Crowding of bronchi in right cardiophrenic angle.

(To face p. 11.

these small sectors of lung substance. This function it can perform because it is rich in blood supply, which increases markedly in response to irritation, thus giving inflammation as the evidence of resistance. Continued inflammation, however, brings much increase in cells which are later permeated by new capillaries from the swollen blood vessels. The result is the formation of fibroblastic cells, which obliterate the new capillaries, so that nature's attempt at repair of the damage leads to interstitial fibrosis. Such repair tissue is fraught with danger to the bronchi and the parenchyma. What was formerly a resilient supporting structure is now a fibroid and restricting band, which impedes the normal expansion and elongation of the bronchi with inspiration. It also interferes with the blood supply of the bronchial artery which runs through it. Further, by its shrinkage it pulls on the surrounding lobules through their supporting septa, with which it is connected. In its advanced stages, such as are seen in bronchiectasis, this traction can actually obliterate alveoli by crushing them. This combined action of constriction of the bronchi and destruction, and consequent collapse of near-by lobules, is well demonstrated in the film of early bronchiectasis in Fig. 20. We can see that the bronchi of the inner bundle of the right lower lobe are crowded together, just outside the lower third of the right cardiac border. Their outlines are congested and enlarged, so that they are much more prominent than normal, and we know there has been destruction of the lobules in their immediate neighbourhood, because we find there is loss of lung markings in the outer lower zone of the lung, where compensatory emphysema has resulted.

Later we shall learn that increase in connective tissue round the bronchi is one cause of the increase in the normal striations on the film, and that it gives characteristic dry crepitations when we listen by stethoscope.

The Supporting Structure of the Lobule

The connecting septa round lobules can be so distended that it breaks down in the disease of emphysema. This can be recognised on the film, as a loss of normal markings; to this point we shall return more fully later in our studies on applied pathology. It can also be broken down by septic material in its blood vessels when this is carried to them as emboli from an infected focus; in this way many small scattered abscesses can appear throughout

the connective tissue over large areas of the lungs in the disease of pyæmia.

Lymphatic Supply

Lymphatics are in abundant supply in the connective tissue round the lobules. Their presence explains the first steps in the pathogenesis of certain lung diseases. The whole system in the lung is divided into two sets, a superficial and a deep, which communicate with each other in the pleura and at the hilum. The superficial set lies in the pleura, which we shall consider later. The deep set runs with every subdivision of the bronchi, the pulmonary artery and the pulmonary veins, and so it follows that the connective tissue between the lobules has an exceedingly rich lymphatic supply. This connective tissue supply is finally linked with lymph spaces that bathe individual acini, and of this fact we must make especial note, as it is by this route that tubercle bacilli finally enter the lung parenchyma in adult phthisis. The bacilli circulate and multiply in the lymph spaces, from which they penetrate the walls of the air-cells.

All lymphoid tissue in the body is meant to be a protecting factor, both at the point of any disease process where it acts as an absorbent agent, and also in its canal system, whereby actual body irritants and their products may be carried to the glands. In other words, the glands are the drains; the lymph channels are the gutters. We see these lymph channels act as a conveyor system in silicosis; they transport dust-containing cells from the alveoli to the lymph spaces round the acini, from there to the connective tissue lymphatics, and thence by peri-vascular and peri-bronchial channels to the lymph glands. Lymphoid tissue carries out the same function in childhood tuberculosis, draining the primary focus to the glands in the hilum. But in adult tuberculosis of the proliferative type we see that it is the lymph-spaces between the alveoli that start the first reaction towards the production of the acinar lesion; not only do the lymph spaces fail to perform their proper function in this case, but they actually break down to become a culture ground for the invading bacilli. All lung lymphoid tissue increases with age. It is the breeding ground for that widespread form of tubercle known as senile phthisis. But it seems to do its utmost to reassert its protective powers. The patient is so little upset constitutionally that he may be a dangerous but unsuspected

carrier of the disease, and is often treated for what he appears to be, a chronic bronchitic.

The Blood Supply

The alveoli are very rich in blood supply, the final capillaries of the pulmonary artery being so numerous as to be a component of their walls, while their connecting and binding septa of connective tissue have an almost equally abundant supply of nutritive vessels, derived from the bronchial artery. The distribution of these arteries explains many points of difficulty in chest diseases, and helps us to understand many of the markings on normal and abnormal skiagrams.

The function of the pulmonary artery is oxygenation of the blood-stream. It originates from the right ventricle, and under the arch of the aorta divides into its right and left branches. At the hilum the right branch lies in front of the right main bronchus, the left branch just above the left main bronchus. It follows, therefore, that the main pulmonary artery and its branches enter in great part into the composition of the hilar shadow on the film. In the lung, each artery follows the bronchi, lying behind and slightly lateral to them, in all their "dichotomous" divisions. They are the guide, as we noted in the last chapter, to the bronchial distribution. We must remember, however, that while close to the hilum the artery and the bronchus are practically the same size, the artery lessens in size very fast as compared with the bronchus, and gives off many more branches within the same distance; this is why when looking at an abnormal film we can tell whether the radial losses of translucency we see in "streaks" are blood vessels or bronchi. Within a distance of an inch, towards the middle of the lung fields, a bronchus might be seen as continuous parallel lines with no branches, while an artery will not so appear. By the time the pulmonary arterial subdivision has reached an acinus it has only about one-fifth of the *diameter of the supplying bronchiole to the alveolus.*

We must remember, however, that the arteries can be materially changed in size by various diseases. Thus in congestive failure, they may be considerably swollen; they then give increased soft markings, generalised throughout the lung fields and lessening towards the peripheries of the film. The opposite effect can be noted in conditions associated with extreme anæmia, where their thin, flaccid tubes are indistinguishable from the normal striations

of connective tissue. These changes will, of course, be accompanied by relative changes in the shape and size of the cardiac shadow, and be apparent in both lung fields.

The function of the bronchial artery is to bring necessary nutrition to the framework of the lung. That for the right lung comes usually from the thoracic aorta, but occasionally from the first or third intercostal artery; the left supply has usually two origins from the thoracic aorta. It is important to remember this difference of origin of the pulmonary and bronchial supplies; the latter as direct from the main arterial vessel has the higher pressure, so a wound or other cause of a break in its wall can prove much more rapidly fatal than the involvement of an arteriole from the pulmonary artery.

As soon as the bronchial arteries enter the lung they enter the connective tissue layer round the bronchi, two or three subdivisions accompanying each bronchial branching. This is why any condition increasing blood supply has the possibilities of marked effects on the bronchial walls: why such conditions of long-standing inflammation as chronic bronchitis and bronchiectasis can cause the havoc of fibrosis round the bronchi affected in the disease. Here we have the explanation for the film of chronic bronchitis. The normal supply from the bronchial artery is very rich. When this supply is swollen by chronic inflammation, and brings the consequent deposition of fibroblasts, we get the evidence on the film in the appearance of parallel lines in the affected area, outlining the larger bronchi in the lower zones of the lung fields. We can find the same effect in other parts of the lung in other diseases—*e.g.*, in the upper zones in chronic pulmonary tuberculosis.

Such rich blood supply is the reason why quite severe hæmorrhage can occur by even small abrasions of the bronchial wall by irritant material inspired into the smallest divisions of the bronchial tree; and why the breaking down of the bronchial wall in bronchiectasis can produce the biggest of all hæmorrhages in lung diseases.

CHAPTER III

THE PLEURA AND THE DIAPHRAGM

THE pleura is in two layers connecting at the hilum. The first, or visceral layer, is closely applied to the lung; it reflects at the hilum to *form* the *second*, or *parietal layer*, which *lines* the *inner* surfaces of the structures internal to the thoracic cage, and the diaphragm. The parietal layer is sensitive. Pain and severe shock can occur when a needle is pushed through it in the induction of artificial pneumothorax, and the pull of adhesions on its inflamed surface may well be the reason for the acute stabbing pain with each respiration in lobar pneumonia.

The two layers have between them a potential space. They do not adhere in health but move easily on each other by virtue of a thin lining of serous fluid, in which the visceral layer can slide on the parietal. In the space there is a pressure which is "negative," in that it is below that of the atmosphere; a needle inserted in this space and connected with a water manometer shows that the pressure varies on an average between -10 cm. of water pressure with inspiration and -5 cm. with expiration. It therefore exerts a constant outward pull on the elastic tissue of the lung, adding to that of the residual air in the alveoli; each inspiration adds to the pull, thus lengthening and opening up the bronchi to receive the inhaled air (see Fig. 10, p. 11).

It follows that any pathological alteration of the negative intrapleural pressure has a marked effect on the elastic tissue of the lung. If it be reduced to the level of atmospheric pressure by air in pneumothorax, or fluid in pleural effusion, the lung must tend to recede to the anchorage of its elastic tissue at the hilum, retracting to a size less than that of complete expiration. The stretch on the elastic tissue of the lung is now maintained only by the residual alveolar air. Raising of the pressure above that of the atmosphere, and therefore above that of residual air, will bring pressure collapse of the alveoli and consequent still further retraction of the lung, for now the pull of the alveoli on the bronchial elastic tissue is being lessened. As we shall see later when discussing the mediastinum, these are the ruling factors in the displacement of mediastinal structures in pleurisy with effusion.

In going round the lung as described, the visceral pleura dips between the lobes, lining the under surface of the one above and the upper surface of that below it. One such lining may appear on a normal postero-anterior film of the chest; it is the lesser fissure between the upper and middle lobes of the right lung, and it shows as a thin, hair-like line running out almost horizontally from the hilum in the third or fourth rib-interspace. Sometimes it casts a double line as the X-rays have cut across it tangentially. Its position is easily recognised when it is forced apart by fluid, or its rich lymphatic supply is inflamed or thickened in such diseases as pneumonia, lung abscess and tuberculosis. If it is seen on the postero-anterior film, its location and direction should be noted carefully, as this may give an indication of a disease process that can be clarified only by further film examination—*e.g.*, lateral film. Thus it is displaced upwards and towards the infra-clavicular zone in collapse of the right upper lobe (see Fig. 8, p. 5).

Fig. 21. Right upper lobe pneumonia.

Other fissures do not show on the normal postero-anterior film, and it may take lateral or lordotic films to show their involvement in pathological conditions.

Some students find difficulty in the interpretation of abnormal films because they forget that the interlobar fissures as they visualise them are but the points of reflection of the pleura, which, in completely covering any lobe must necessarily cover, by the visceral layer, the lessening triangular wedge, known as the lingula. The shadow cast by normal lung being but the superimposed shadows of its varying structures from front to back on the postero-anterior film, it follows that density of shadow in developed lobar

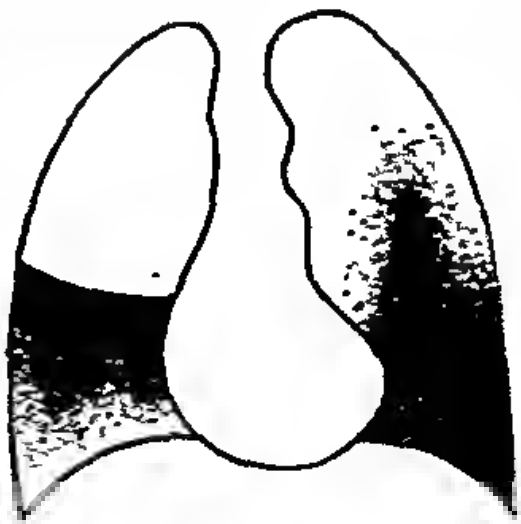

Fig. 22. Pneumonia of the right middle lobe, and of the left lower lobe.

pneumonia varies by depth of tissue, and not by intensity of disease, which is equal in the body and the lingula of the affected lobe. We *cannot expect the shadow of right upper* lobe pneumonia to be as dense in its lower limits as in its upper because the lobe thins out considerably as it goes downwards; we recognise middle lobe pneumonia because it has a dense shadow in the right mid-zone which lessens steadily towards the base, so that through its lower limits we can see the right diaphragm (see Figs. 21 and 22).

This explains, too, why collapsing lobes cast characteristic shadows; why the lesser interlobar fissure throws a heavy density upwards and outwards as the lingula recedes on to it in right upper lobe collapse (see Fig. 8, p. 5). Likewise we realise that when a right lower lobe collapses, the lingula has already shrunk downwards a long way before we see on the film the upper limit of the lobe defined by a line running downwards and outwards towards the costo-phrenic angle from a point at the upper edge of the right auricle on the right side of the cardiac shadow (see Fig. 15, p. 8, and Fig. 72, p. 90). The position of these fissures in the lateral film is discussed in Chapter V.

With such changes in shape of lobes we shall learn to correlate changes in the normal striations that appear on the film by shadows of vascular supply and supporting structure. Thus we have already noted that in right upper lobe collapse we can no longer see the three normal branchings of the pulmonary artery which supply it, while in the rest of the lung field we shall find, spread out much more horizontally than normal, the blood vessels of the middle and lower lobes, which have been blown out by complementary emphysema to occupy the space vacated by the shrunk upper lobe. *In this emphysematous area* we shall at the same time find much increased translucency as against the normal, because the supporting structure of the vesicles is considerably stretched. We therefore make our diagnosis by *noting two opposite processes: increased density and increased* translucency; one with loss of blood supply, the other with spread of blood supply and of supporting structure. In other words, where we suspect a change in one type of lung striation we must look for a corresponding change in the other, and if we fail to find one in the presence of another on a postero-anterior film we must make other investigations, such as the taking of lateral films or the injection of lipiodol. Thus in complete collapse of

the left lower lobe we may see on the postero-anterior film only the gross emphysema of the upper lobe filling the hemi-thorax; we may not be seeing the collapsed lobe because it is completely hidden by the cardiac shadow: films taken in the postero-anterior and lateral positions before and after the injection of lipiodol will aid in the diagnosis.

The Diaphragm

The diaphragm is the most important muscle in respiration and therefore, next to the heart, the most important muscle for the maintenance of life. It consists of a thin, movable partition lying between the thorax and the abdominal contents. It is convex upwards, its right half being slightly higher than its left and taking a position just below the nipple at respiratory rest. There is a central, tendinous, very strong portion and three fleshy portions which come from the ribs, the sternum, and the spine, and are therefore named costal, sternal and vertebral. Of these we must note especially the costal; its origins are from the inner surfaces of the lower six costal cartilages on each side. When the diaphragm is depressed they may be seen on a postero-anterior film in their diaphragmatic insertions, as depressions between convexities, thus forming a series of waves, and such a picture, while commoner in emphysema, does not necessarily connote any disease process. They are sometimes wrongly interpreted as evidence of adhesions or of old pleurisy (see Fig. 23). If there is really adhesion of the diaphragm there will be no clear outline, but a merging of lung shadows into the deeper density of the muscle.

FIG. 23. The costal insertions of the diaphragm.

These normal alterations in the dome may also be confused with the very common finding of a tent-like shadow that sits on the diaphragm about its middle third. This is the end-result of a pleural reaction in the main fissure (see Fig. 32, p. 34).

The central tendinous portion may appear as a hump on the middle of the diaphragm: it is without clinical significance, but may require investigation against the comparatively rare pleural growth that can develop at this point.

By the contraction of its fleshy portions, through the action of

the phrenic nerve, the diaphragm becomes less convex, and so increases the vertical diameter of the thorax in inspiration. In ordinary breathing this range of movement is about three-quarters of an inch. and in every examination of the chest it should be looked for by percussion of the lung bases, posteriorly and in the axillæ, since deep inspiration should move the diaphragm downwards by at least a finger's breadth, as shown by the higher pitch of descending air-containing lung.

It follows that interference with the phrenic nerve, at any point throughout its course over the cupola and past the hilum to the insertion of its fibres, will cause rise of the diaphragm. Evidence of this is common nowadays in the films of patients in whom the nerve has been crushed, or evulsed, as a treatment for pulmonary tuberculosis. Such films demonstrate that the diaphragm, as it moves downwards in inspiration, exerts a distinct pull on the apex of the lung, for when it is immobilised an apical tuberculous lesion shrinks, and gets the rest it requires to aid healing.

Adhesion between its pleural lining and the visceral pleura of the lobes in contact with it will fix the diaphragm, while emphysema will depress and flatten it. Contraction of the lung by loss of elasticity will cause it to rise, *e.g.*, in collapse of the right middle lobe (see Fig. 63, p. 88). The recognition of such changes in its position and movement by physical and film examination can therefore be of very distinct help in the diagnosis of underlying lung disease.

CHAPTER IV

LUNG AND MEDIASTINAL MOVEMENT IN HEALTH AND DISEASE

THE mediastinum is a space running antero-posteriorly in the mid-line of the thorax, between the sternum and the spine. It shows on the postero-anterior film as the shadow of parts of its contents, so that we can identify the trachea to a point just above its bifurcation, the outline of the cardiac shadow, the aortic notch, and the structures that make up each hilum. As we have already noted, all these mediastinal structures are embedded in areolar tissue, which covers and binds them together, but yet is elastic in composition, so that it can move with systole and diastole of the heart and the great blood vessels.

We have already noted too that it is connected by the layer of pretracheal fascia with the investing fascia of the structures in the neck, and that thereby its displacement to one or other side of its central position is reflected to an increased tension of the tendinous portion of the sternomastoid muscle on the same side.

Again, we saw that the mediastinum has a weak point, on which it swings as on a hinge, and that the trachea can move laterally both above and below this hinge; for example, that it can be definitely kinked by disease localised in the parenchyma below the collar bone. The heart is displaced much less readily. A much heavier structure, it has its greatest weight in the lower mediastinum, so that it is moved laterally only by disease conditions in the lower half of the thorax or throughout one whole side of the thorax. Thus the apex beat is seldom abnormal in position in tuberculosis of one upper lobe, but it can be moved quite markedly to the opposite side in the presence of a pleural effusion and to the same side in post-pneumonic fibrosis, this being a disease which commonly affects the whole of one lung field.

An important point to note is that the heart will swing round its long axis much more readily than it will move laterally. This explains the anomalous outline it presents in the normal abnormality of scoliosis, which has its usual concavity to the left. in lying into this hollow the heart swings on its long axis, its

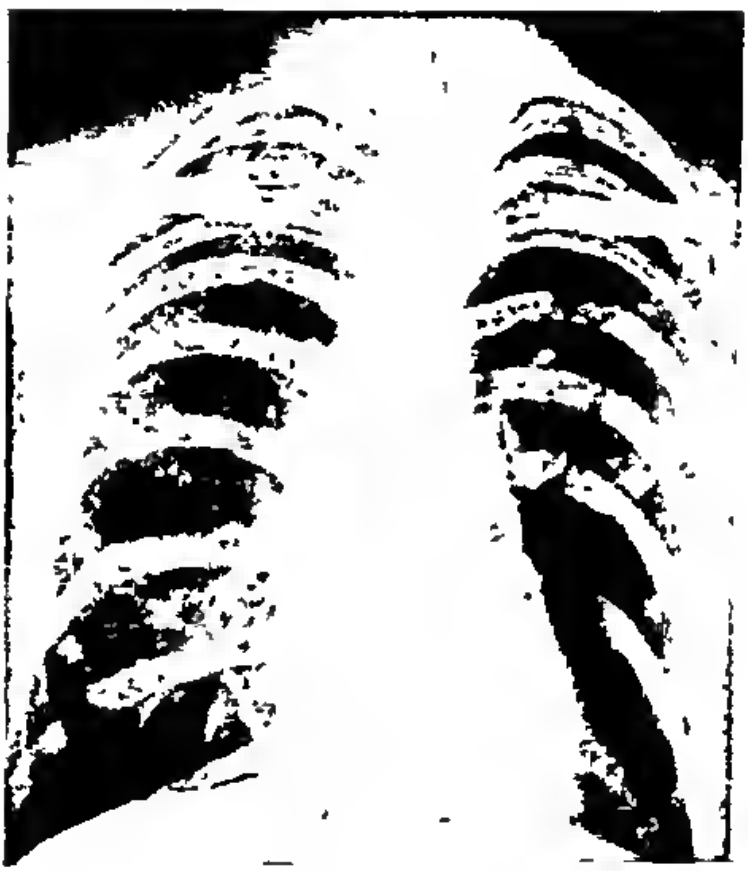

FIG. 24. Marked scoliosis with convexity to the right.

[To face p. 23.

right border receding and its left border coming forward, so bringing the pulmonary conus on the left side into undue prominence, and uncovering the right hilar region (Fig. 24). On the postero-anterior film there is therefore a filling in of the normal triangle of translucency that has its apex between the aortic notch and the normal prominence of the conus, giving an outline that simulates that of mitral stenosis on the left side, while the shadows of the right hilum and of the branches of the pulmonary artery to the right lower lobe stand out prominently. Such abnormalities might be read as evidence of disease if we did not note the accompanying widening of the right lower rib-interspaces and the compensating fall-in of the left lower rib-interspaces. We can see too why it is, by this tendency to swing on its long axis, that the position of the heart at respiratory rest may mislead us in such a disease as collapse of the right lower lobe. As it shrinks, from such a cause as bronchial carcinoma, the lobe goes downwards, inwards and backwards, and causes the heart to swing on its long axis so that the right lower border recedes towards the spine, the left lower border comes forward. Later the whole heart goes backwards. If we have to rely on physical signs for our diagnosis we shall often find more help towards the opinion that the mediastinum is affected by noting the tense right sterno-mastoid than by concluding anything from the position of the apex beat. If we can watch the patient on the screen, however, we shall see the heart move laterally and bodily to the right, in its lower half, with each inspiration, and come back to its former position with expiration. This is because the intra-pleural pressure becomes more negative at each inspiration, and drags the whole mediastinum towards the periphery.

A little later we shall see why the heart goes backwards in collapse of the right or left lower lobe.

Experience shows that mobility of mediastinal structures varies considerably in different individuals. The pressure in the pleural cavity in two subjects may be the same as judged by the same manometer in conditions as nearly equal as we can make them, yet the amount of displacement from normal in disease of seemingly equal extent may be entirely different. In one, a small pneumothorax can cause great bulging of the anterior mediastinum . in another it will hardly move this area. This means that the mediastinum is never a fixed and invariable entity , we cannot by working backwards give an infallible estimate of the amount

of lung disease by basing it on the amount of mediastinal shift, and all we can say is that some damage has taken place. On the other hand, we must realise that disease of the mediastinum can have a definite stiffening effect; thickening of the mediastinal pleura can combat peripheral changes of pressure. Thus we may get much more falling in of ribs and raising of the diaphragm than we would expect from the amount of damage evident in the parenchyma.

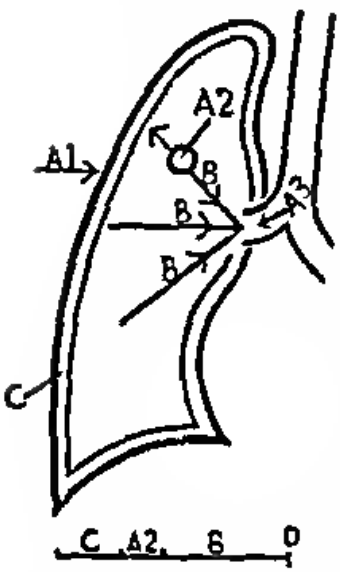

FIG 25. The forces acting on the mediastinum.
A_1. Air pressure on chest wall.
A_2. Air in alveolus.
A_3. Air inspired into bronchus.
B. Elastic tissue.
C. Intra-pleural pressure.
D. Mediastinum.

The mediastinum must always be envisaged as one entity, not as something in two halves, one for each hemi-thorax. It is one central elastic structure, both maintaining and being maintained by one intra-thoracic pressure. If there is a fall of pressure on one side of the thorax, there is an immediate, automatic rise on the other. Fibrosis on the right implies emphysema on the left. Now we can understand why the heart goes backwards in right lower lobe collapse. The pressure in the right lower mediastinum is lessened; it cannot put up normal resistance to the pressure in the left lower mediastinum, with the result that the left lower lobe moves across the front of the heart and pushes it backwards.

We are now in a position to sum up our considerations of applied anatomy, to discuss the action of the various lung and intra-pleural forces on the mediastinum in health, and see how they react on each other and on this central elastic structure in disease.

In the accompanying diagram (Fig. 25), "D" represents the mediastinum. "A" represents atmospheric pressure, which acts in three ways:—

(1) As A_1, pressing in on the chest wall.

(2) As A_2, the pressure of residual air in the alveoli; we have seen that this is maintained by the bag-pipe action of the smooth muscles of the bronchi. A_2 acts in every direction throughout the lung fields, pressing against the periphery, the diaphragm and the mediastinum. We

have seen it demonstrate its force against the mediastinum in collapse of the right lower lobe. It also presses on surrounding alveoli, so if it is lost in any area the surrounding alveoli immediately distend, giving localised emphysema.

(3) As A_3, the air entering by the main bronchus.

"B" represents the elastic tissue of the lung, which runs with every bronchial division from the hilum to the alveoli. The more a piece of elastic is stretched, the more it tries to contract to its fixed point. As lung elastic tissue is on constant stretch by the action of the residual air in the alveoli (A_2), it is always attempting to pull the lung inwards to its anchorage at the hilum, where it is continuous with the elastic tissue of the trachea. Therefore in proportion with the destruction of the elastic tissue the lung will always *contract towards the hilum.*

"C" represents the sub-atmospheric, "negative" pressure in the pleural cavity. This exerts a steady outward pull on the lung, and *is increased with each inspiration.*

Again we must remind ourselves that all these forces are acting in exactly the opposite direction against the mediastinum on the opposite side. A_2 on the right is pushing to the left; A_2 on the left is pushing to the right. Thus the central balanced position of the mediastinal structures.

Let us now consider examples of disease conditions affecting each of these factors and see how they react on the mediastinum.

A_1 is a constant factor in all ordinary conditions of life in health and disease.

A_2 may alter by such conditions as:—

(1) blockage of the bronchus, through carcinoma, or septic or caseated material.

(2) destruction of the alveoli, as in tuberculosis of adult type.

There is therefore loss of residual air. A_1 has less resistance offered to it, so the hemi-thorax tends to shrink and the ribs tend to fall in. *There is less resistance to the diaphragm, so it* will rise higher at rest and with complete expiration. There is less resistance to neighbouring alveoli and so they show emphysema. There is less resistance to the mediastinum, so the force on the opposite side will push the mediastinum to the diseased side. Finally there is less pull on the elastic tissue anchored to the trachea through the main bronchus and so the lung will retract to the hilum, and this means that "C" is now much less

opposed and drags the whole lung outwards. We saw an extreme example of this action of "C" in the detailed discussions of what happens in collapse of the right lower lobe. We noted that the lower half of the mediastinum, including the lower half of the heart, swings out to the right with each inspiration, and comes back to more central position with each expiration.

We can represent this in a simple line drawing (see Fig. 25), remembering that all processes are acting equally in the reverse direction in the opposite hemi-thorax.

A_2 is destroyed. B therefore shrinks towards D, and C, now unopposed, pulls B and D outwards. The mediastinum therefore drifts to the side of the atelectasis, the amount depending on how much of A_2 is destroyed. We see now why it is that we get a positive sternomastoid sign with loss of residual air. It is not due directly to the collapsed tissue, but to such lung tissue as remains unaffected and is dragged over bodily by the intra-pleural pressure. Its drag is communicated to the trachea.

If we have followed these arguments we can see why such diseases as unilateral tuberculosis or bronchiectasis must displace the mediastinum to the side of the lesion.

We have here too an explanation of cavity formation. There is marked localised loss of elastic tissue by organisation and collapse of lobules, and therefore loss of the pressure of their residual air. Immediately surrounding lobules are unopposed in their own inherent pressure, and blow out to become emphysematous, and so their weakened walls are dragged in every direction towards the periphery, both by those external to them and by the intra-pleural pressure. Apart altogether from any extension of the actual disease process there is physical force behind the production of increasing cavitation.

It is possible to follow now why diseases which replace residual air by material which keeps the alveolar walls distended cause no displacement of the mediastinum, unless they overfill the vesicles and so pull the mediastinum a little towards them. Pneumonia fills the alveoli with exudate; they cannot move, but they keep the elastic tissue of the lung on stretch as full inspiration does. Therefore, uncomplicated lobar pneumonia does not give a sternomastoid sign, nor does an uncomplicated abscess of lung, which, as we shall discuss later, is merely a filling of lobules by inspired septic material. Tuberculosis, on the other hand, will do so; it destroys the alveoli. Collapse of the lung does; it takes away

residual air. We have therefore here a very distinct aid towards differential diagnosis.

Let us now consider disease conditions affecting C, the sub-atmospheric, negative, intra-pleural pressure. If we have a case of adhesive pleurisy C disappears. A_2 now attempts to fill the space vacated by C, and drags B, and therefore D, with it; we get a positive sternomastoid sign on the same side as the disease.

If fluid or air enters the pleural space to remove the negative pressure and bring it to zero, A_2 alone maintains stretch on B: the lung retracts to the hilum and is not moved outwards by inspiration. But the force in the opposite pleural cavity is correspondingly greater and so we have a drag to this opposite side, on which the sternomastoid sign will be positive. In other words, it does not require actual push from the right, in a right-sided pleural effusion, to produce mediastinal displacement to the left.

If the intra-pleural pressure rises above atmospheric, by increasing air or fluid, it will be greater than A_2. The lung is now pushed in by pressure collapse. The outward pull of A_2 is lost to B which retracts still more towards D. Thus fluid in the right pleural cavity pushes on the mediastinum, and at the same time the pull from the left side is still more strengthened and we have now a very marked left sternomastoid tension.

Much will depend now on the condition of B. If there is much fibrosis of lung opposing the elasticity of B, the lung will not retract *to the mediastinum, D, but only* more towards D *if it is* pushed over *bodily*. Thus a pneumothorax which complicates established pulmonary tuberculosis can give much more displacement of the mediastinum than a simple non-tuberculous pneumothorax which is due to rupture of a bulla. The lung without disease collapses quite readily.

If we follow this explanation of lung and mediastinal movement we shall be in a better position to understand the physical and X-ray signs of those commoner chest diseases whose pathological changes are considered in Section IV.

CHAPTER V

THE NORMAL CHEST FILM

A FULL understanding of what constitutes a normal film of the chest is a necessity for the student who hopes to interpret the abnormal. The following description relates only to the points on a postero-anterior and a lateral film which concern the average practising physician. It does not go into all the finer detail which concerns the specialist chest physician, the cardiologist or the pure radiologist, which detail the student may find in specialised text-books. It tries to set forth only those points which the physician can correlate with his physical signs and their causative pathology, so that he can go beyond the merely objective reading of abnormal lung shadows.

Some preliminary observations on prime principles must be set out. Thus we have already noted that a film is merely a composite photograph of superimposed shadows of various structures through which the rays pass. With shadows of disease at any one depth we shall therefore see the shadows of normal structures in front of or behind them. An early "adult" tuberculous focus is completely homogeneous to tomograph examination, and much more defined on an antero-posterior than on a postero-anterior film. On this last film it may appear to be striated, because, being a posterior lesion, it is shown with much normal lung in front of it, and such normal lung will now throw its striations in greater comparative relief against the background of the disease focus. In the same way we shall see varying shadows of a broncho-pulmonary segment in the lung fields. If it be supplied by a laterally directed bronchiole this cone of tissue will appear on the postero-anterior film as a triangle with its apex at the point of entry of the bronchiole; if it be supplied by a bronchiole that goes dorsally, it will appear circular or oval as photographed along a line between its apex and the centre of its circular base (see Fig. 12, p. 7, and Fig. 43, p. 61).

A good film is the result of good positioning, proper exposure as judged by muscular development of the subject, and good processing. Although the result is by no means a stereoscopic picture, there is no doubt in the mind of the writer that viewing such a film first at close quarters, and then at a distance of some

5 feet, will tell the observer much of the composition of abnormal shadows, if not of their depth in the lung fields.

Of late years medical literature has shown much less variation in the objective and interpretative readings of chest disease, because certain criteria of a good postero-anterior film have been more or less generally accepted. These are that it may show the lesser fissure of the right lung in the third or fourth interspace, that it should show the superior vena cava with a density slightly less than that of the heart, and that it must show the shadows of the branches of the pulmonary arteries radiating from the hila throughout some three-quarters of the lung fields.

It must be borne in mind that the hilar shadows are themselves predominantly vascular. As the pulmonary arteries and veins give the main part of the rather indefinite semi-circular opacity on each side of the cardiac shadow, it follows that this opacity will be increased by all conditions characterised by hyperæmia. Seeing the left one is already denser by the prominence of the conus in the normal abnormality of scoliosis, such a condition as broncho-pneumonia in a scoliotic subject will produce very heavy left hilar density, both during the acute stage of the disease and for long after clinical recovery.

The Normal Postero-anterior Film

The first lesson the student must learn is to read every film, normal or abnormal, on a definite pre-arranged plan. If this is not done consistently even the expert observer may find his eye has been arrested by some striking area of density or contrast, or by one interesting detail in one area of the lung fields, and that he has missed an abnormality of lesser extent or contrast that is nevertheless of the first importance in diagnosis. For example, a filling in of one costo-phrenic angle is dependent for the interpretation of its real significance on the presence or absence of a lung focus. We have already noted how a grave mistake may be made in the interpretation of the film of a simple scoliosis if we do not connect the variations in the heart outline and the right hilar shadows with the changes in size of the lower rib interspaces.

A simple plan is to read every postero-anterior film under the following seven headings :—

(1) The thoracic cage.

(2) The trachea ; shape and position.

(3) The heart ; size, shape and position.
(4) The diaphragm ; position and contour.
(5) The upper zones of the lung fields.
(6) The middle zones.
(7) The lower zones ; comparing the right with the left in each case.

The Thoracic Cage

The first thing the student of chest films should learn is to recognise the shadows of the bony thorax, and those of muscle, fascia and skin that can appear on a normal film.

Fig. 27. To show how tuberculous foci may be hidden by rib shadows.

The ribs are seen more clearly or less clearly than normal in certain pleural and lung diseases. Tuberculous pleural effusion displaces the lung shadows inwards ; through the even, light loss of translucency cast by the fluid in the periphery the ribs show up more clearly than normal (see Fig. 5, p. 4). They are still more prominent in the film of pneumothorax. In emphysema and pneumonia they are less prominent, owing to the dense shadows thrown by these diseases.

The anterior ends of the ribs often continue into the shadow of calcification of cartilage, which is more dense, and less even, so that it can simulate the shadow cast by pulmonary disease, especially tuberculosis. Usually the condition is seen on more than one rib, although it is commonest in the first rib. Even then it is almost always bilateral, and it is a fairly safe aid to remember that lung lesions are rarely similar in position and characteristics on both sides of the chest.

Fusion and bifurcation of the anterior ends of ribs is common. Special films may be necessary to discriminate between their shadows and those of underlying lung disease. The commonest of all these "normal abnormalities" is fusion of the anterior ends of the first and second ribs (see Figs. 26 and 29).

Points of intersection and overlap of rib-shadows can cause considerable difficulty (see Fig. 27). This is especially so at the apices, where the shadows of the clavicle, of the sterno-mastoid muscle, and of skin and fascial folds, complicate the

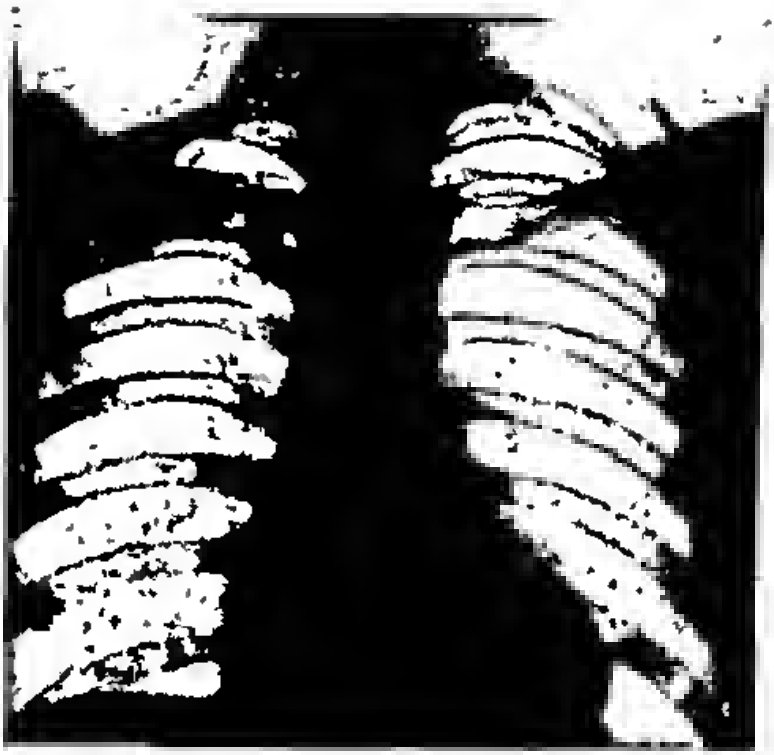

Fig. 26. Fusion of anterior ends of first and second right ribs.

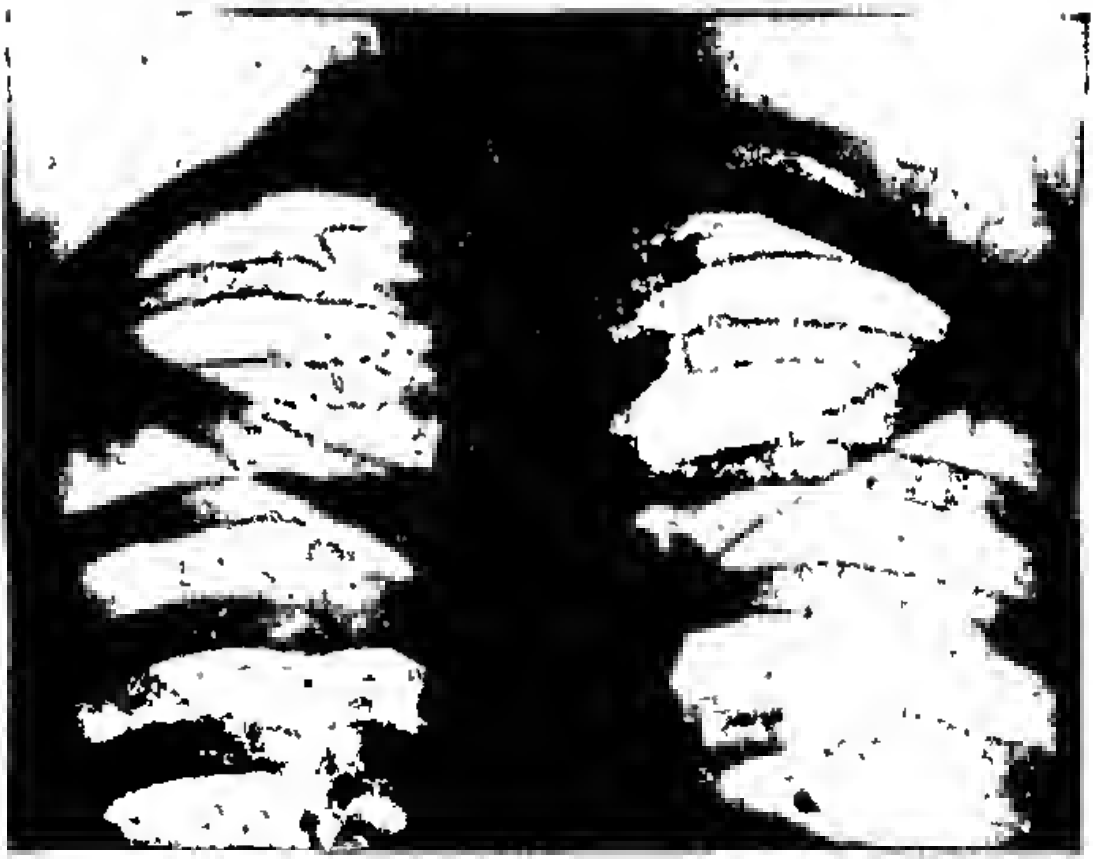

Fig. 29. On the right an under-developed first rib. On the left a ball-and-socket junction of first and second ribs

(To face p. 2)

picture. Fig. 28 illustrates how these shadows may be recognised That cast by the sternomastoid muscle may need careful scrutiny as it lies in the area involved in tuberculosis arising in and around the posterior apical branch of the upper lobe bronchus.

Bad positioning causes the shadows of the scapulæ to obscure the outer parts of the lung fields, but these should be recognised as readily as the rounded shadows due to the breasts in the lower

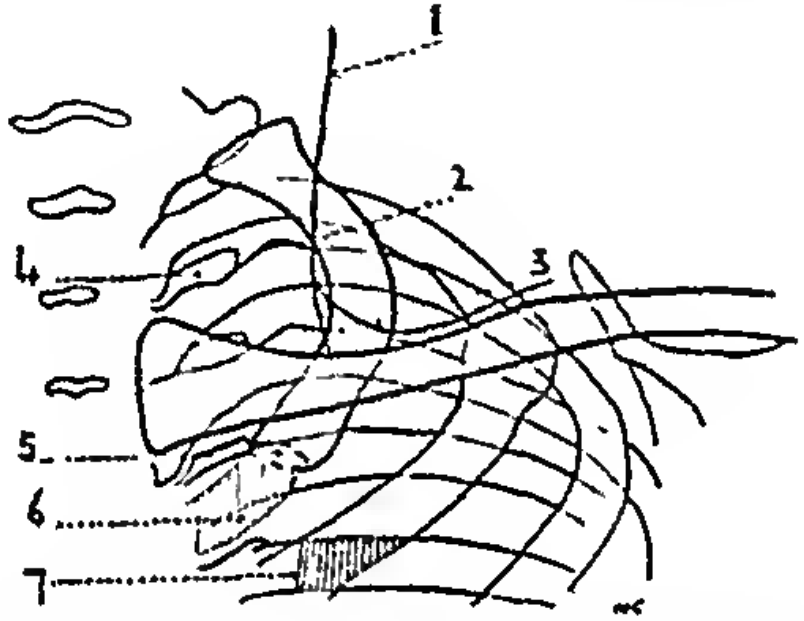

FIG. 28. Drawing to show normal shadows at the apex which may cause difficulty in reading miniature films. 1 = Sternomastoid muscle. 2 = Costal tubercle of third rib. 3 = Shadow due to skin and fascia. 4 = Transverse process of vertebra. 5 = Tip of first rib. 6 = Calcification of first costal cartilage. 7 = Area of density due to superimposition of the shadows of two ribs crossing one another. Note also the expansion shown on the course of the second rib as it crosses the clavicle.

zones in females. The shadows of the pectoral muscles and their blood supply may give quite pronounced striations and loss of translucency in the infra-clavicular and mid-zones of the lung fields, especially on the right side, but generally in this case the lower edge of the pectoralis can be seen as a sharp line running downwards and inwards.

The Trachea

The trachea should be seen as a band in front of the vertebræ, slightly inclined to the right, and with no clearly defined walls. It may be a little to one side in scoliosis, and is generally seen as far as its bifurcation at the level of the fifth dorsal vertebra.

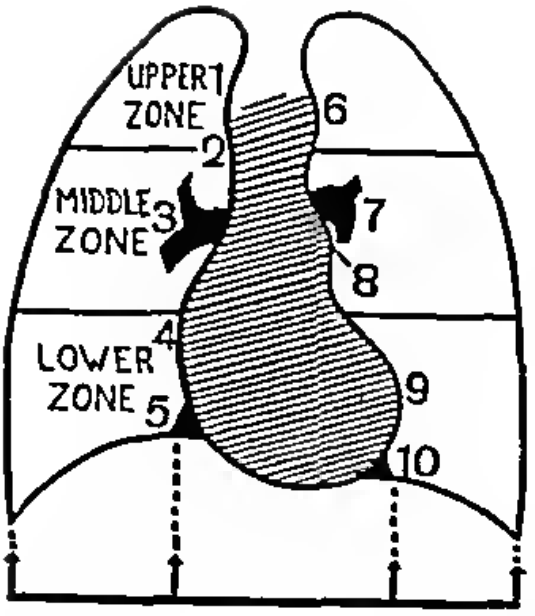

FIG. 30. Points to be noted in reading the postero-anterior film of the chest.

The Heart (see Fig. 30)

An understanding of the normal cardiac outline is a necessity, since its alteration accompanies, and can aid in the diagnosis of, diseases which are primarily pulmonary in origin. The following description does not give details required for the study of primary cardiac disease.

The cardiac outline shows as an opaque pear-shaped shadow. The transverse diameter is of no pathological significance unless the heart be of the longitudinal type; it cannot help where it is of the squat, transverse type (see Fig. 31). In this latter case the outline of the left lower border is usually indefinite, as the heart is pushed upwards by the diaphragm. The student should learn to recognise that the shadow of such a heart lessens steadily from its mid-point outwards, and note that under its indefinite inferior border he can still see the density of the diaphragm all the way to the cardio-phrenic angle. He will then have no fear of confusing it with the characteristic shadow of lower lobe collapse (compare Figs. 72 and 73, p. 90). Here there will be dense loss of translucency filling the cardiophrenic angle; the opacity is equal to that of the mid-point of the cardiac shadow with which it is continuous. Its outer border is sharply defined. The diaphragm does not show as a definite linear outline below it, while the costo-phrenic angle is abnormally free from striations because it is filled by the emphysematous upper lobe.

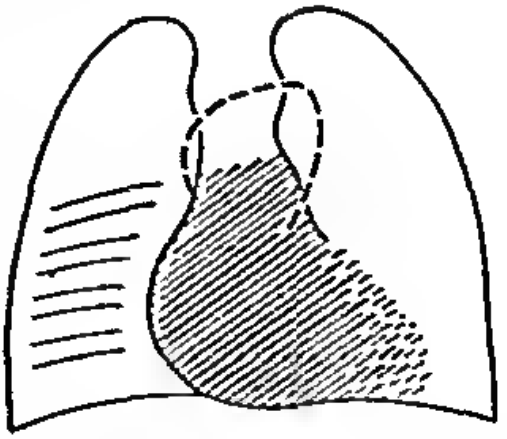

FIG. 31. Chronic bronchitis and emphysema showing squat heart, unfolded aorta, splaying of the ribs and flattening of the diaphragm.

Normal hilar glands do not show on a postero-anterior film; on the other hand, it is usually unwise to diagnose them as enlarged unless they are undoubtedly very prominent, for we have already noted that the hilum shadow is cast mainly by blood vessels, which can enlarge considerably in all conditions giving hyperæmia. While crenated calcified glands should be noted, and, when they are seen, a primary Ghon's focus of childhood tuberculosis should be searched for in the lungs, their finding should not be considered as evidence of present disease. They do not constitute so-called "hilum tuberculosis," for there is no such primary disease; indeed they are so common that, like scoliosis, they should be considered a normal abnormality in the film of an adult.

The Diaphragm

The diaphragm appears as two clear cut, dome-shaped shadows, one on either side of the cardiac shadow, acting as the lower limits to the basal translucency of the lung-fields. The right, which rises to the sixth interspace, is a little higher than the left, usually to the extent of one interspace. The blood vessel shadows may be seen to continue below it, but neither in health nor disease, unless they are injected with lipiodol, do we see the bronchi in this position.

Fig. 32. Showing azygos lobe, blood vessels and bronchi end-on, tenting of the diaphragm and obliteration of the costo-phrenic angle.

There are two common findings that must not be read as evidence of abnormality of clinical import (Fig. 32). The first is so-called "tenting" of the diaphragm. A small triangle, of short base, sitting on the diaphragm, and drawn into a rather elongated apex, is seen towards the inner side of the mid-point of one or other half of the muscle. This is a slight pleural reaction in the main fissure, and is a common finding with broncho-pneumonia even when, by the history, this condition has been of so low-grade that it has not upset the patient sufficiently to make him seek advice. It remains for a long time, even permanently. It must not be read as an adhesion or as evidence of lung disease, unless a definite pulmonary focus is seen, when it may be due to contraction of the fibres of the phrenic

FIG 33. The azygos lobe

[To face p. 35.

nerve, which has been involved in a disease process in some part of its course over the cupola and past the hilum. Such peaking is never then at the position of the main fissure, and its commonest cause is apical fibrosis, usually tuberculous in origin.

The second is a filling in of the costo-phrenic angle on one or other side by an opaque shadow. This is again a common sequence of a previous pneumonia or broncho-pneumonia; the exudate on the visceral layer of the pleura has organised. It is not evidence of present lung disease, and of no significance unless a lung focus is seen with it, in which case it is usually evidence of a secondary pleural reaction to a tuberculous deposit.

The Zones of the Lung Fields

The zones of the lung fields are defined in accordance with the diagram (Fig. 30).

The upper zones are the areas lying between the upper apices and a line drawn horizontally across the anterior lower ends of the second ribs.

The middle zones lie between *this line* and *one drawn similarly* across the anterior lower ends of the fourth ribs.

The lower zones lie between this second horizontal line and the lung bases.

These zones are used to designate the position of any abnormality noted on the film. Each zone should be compared carefully with its fellow on the opposite side before any condition noted is judged as a definite departure from the normal. It has already been noted that disease processes are never exactly repeated in opposite zones, and that if this simple precaution is not employed it is possible for the common condition of calcification of the first costal cartilage to be interpreted as a tuberculous focus.

Normal lungs appear as dark areas with many linear markings throughout them, due to the shadows of the main pulmonary arterial branches and to supporting structure of the parenchyma. We have seen that the hair-like line of the lesser fissure can appear in the third or fourth rib interspace; no other part of the pleura or its fissures should appear in an absolutely normal film, although it is not unusual to find an azygos lobe in the right upper zone (see Figs. 32 and 33). Here a fine line divides the right cupola by running downwards and inwards to the upper part of the cardiac border, at which point there may appear a well-

marked, but small oval opacity which is the azygos vein, round which the visceral pleura has dipped during development. This lobe has a slight loss of translucency, as it contains less residual air than the outer part of the upper lobe.

We have already noted that the radiations due to the pulmonary arterial supply go outwards from each hilum, which is itself due in most part of its confused shadow to the main arteries and the pulmonary veins. Along their course, and also lessening steadily towards the periphery, we may see round opaque shadows of blood vessels caught end-on by the rays as they go backwards. In the same way, while we do not see normal bronchi, which go laterally, we can see the round well-defined circles with clear centres which are the shadows cast by backward-going bronchioles (see Fig. 32). They may be of fair size near the hilum, and should not be read as cavities or mistaken for primary foci of "childhood" tubercle, which are opaque, irregular and hard-looking, and usually well out towards the periphery.

At the danger of repetition, it must be stressed that variations in the normal striations are as much evidence of abnormality as the shadow of the disease process which causes their variations. If we see in the outer part of the right lower zone the shadow of pleural effusion, we must look carefully at the striations in the mid and inner parts of this zone. If the lung has no disease in it we expect to see heavier striations caused by hyperæmia from the pressure collapse of the parenchyma; if we can see no vessel markings we know we are dealing with a pleural effusion which is secondary to a consolidation of the lower lobe.

The Lateral Film (see Figs. 34A and 34B.)

A lateral film may be taken to give additional information in the localisation of lung disease. For example it will throw a shadow that appears to be "hilar" into its true position in the apex of the lower lobe in the case of abscess formation in that broncho-pulmonary segment which is supplied by the posterior horizontal branch of the right lower lobe bronchus.

Lateral films may be taken of the right and left lung fields; it must be realised that the shadows of the opposite side, although out of focus, always contribute to the picture. In the accompanying diagrammatic representation of the left lateral film there can be seen between the vertebral column and the sternum :—

(1) The diaphragm.

(2) The posterior recess of the costo-phrenic angle.
(3) The cardiac shadow resting on the anterior surface of the diaphragm, and continuous in its upper limit with the arch of the aorta.

The position of the main interlobar fissure is shown as a line (4) running from a point below and posterior to the apex downwards

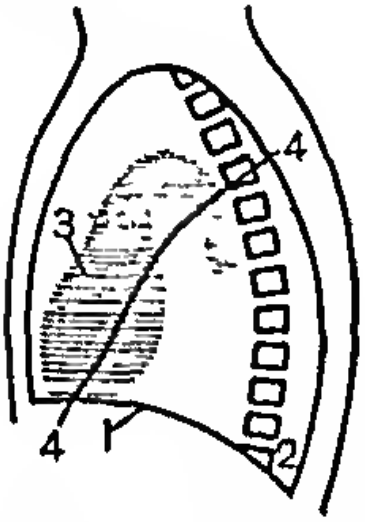

FIG. 31A. The left lateral film.

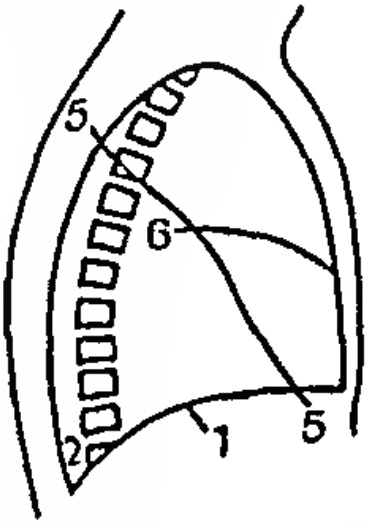

FIG. 31B. The right lateral film.

and forwards to a point near the junction of the middle and anterior thirds of the diaphragm. The lower lobe therefore occupies the area below this line.

On the diagram of the right lateral film the position of the greater fissure is shown as the line marked (5), while in addition the lesser fissure is shown as (6), running between the hilum and the posterior border of the sternum. It may appear on the lateral, as it does on the posterior-anterior film, in the absolutely normal subject.

SECTION II

CHAPTER VI

PHYSICAL EXAMINATION—INSPECTION, PALPATION AND PERCUSSION

PHYSICAL examination of the chest has suffered severe criticism in the past few years. This is due mainly to the realisation among tuberculosis workers that phthisis can exist for months, and even years, without producing symptoms, and that in its earlier stages it may give no abnormal findings to the most expert examiner, while showing definite abnormal shadows on the film. The introduction of mass radiography was urged for these reasons; its arrival has accentuated the criticism. Nevertheless, physical examination, like bacteriological examination, must remain for ultimate diagnosis. The X-ray is not the whole patient. In the hands of an expert it can give detail of diagnosis with uncanny accuracy, but without serial examinations it can seldom give the answer on activity or retrogression of the disease process. The clinical condition as a whole, part of which can be summated only by expert physical examination, can alone lead the physician to an opinion on the present pathogenesis, on which he must base his immediate treatment.

Nor must we confine physical examination to any one of its parts. "Eyes first, hands next, and ears last" is still a good guide. It is the writer's experience that many students concentrate too much on the stethoscope, and that the statement that there are no physical signs means usually that the examiner has found nothing abnormal to auscultation.

It will be assumed that the student is familiar with all the usual methods; only such necessary aids, and such points as the writer has found of help to post-graduates, will be detailed.

(*a*) **Clubbing of the Fingers.** Inspection of the terminal phalanges should be made in all patients suspected of chest diseases, acute or chronic. "Clubbing" is the phalangeal form of hypertrophic pulmonary osteo-arthropathy, which is caused by the forming of a thin subperiosteal layer of bone, and thickening of connective tissue over it. It is found in chronic heart affections and chronic jaundice, but is most commonly associated with lung

diseases. Toxins and chronic congestion are said to have something to do with it, but its real cause is unknown; we do not know why in the terminal phalanges it shows such marked thickening of the soft tissues in some cases which have no apparent difference in their lung pathology as against their colleagues with no finger changes. Its absence, then, is of no diagnostic significance; its particular type when present can be so characteristic as to be diagnostic between "septic" and "fibroid" diseases; between, *e.g.*, lung abscess and pulmonary tuberculosis.

The two main types show a first stage in common. This consists in a filling in of the connective tissue at the root of the nail, so that there is no gap between the skin fold and the proximal end of the nail such as appears on the average normal finger. In addition, this excess tissue, slightly raised, and pushing up the skin fold, is always cyanotic and glistening, as if by continual polishing. While this initial change is later in appearance in phthisis than in the acute septic conditions, where, as in interlobar empyema complicating pneumonia it can appear within a few days of the onset, it can be very helpful as a hint of the possible presence of a symptomless phthisis.

In the further changes associated with proliferative tubercle of adult type the nail becomes humped, bowed dorsally, smooth and rather pearly-pink, while the palmar surface of the phalanx becomes more and more pendulous and baggy. The finger is thus given a claw-like appearance and may even look tapering, as the dorsal-palmar alteration is not offset by any marked change in the lateral dimension.

This is entirely unlike the effect of septic disease, either acute or chronic, as in lung abscess and bronchiectasis, where the alteration is generalised over the phalanx. This alteration is in all dimensions, gross and "drumstick" in effect, often bulging sharply at the joint with the proximal phalanx, to stand out as an ugly blunt stump. It can come on so rapidly in acute sepsis as to be noted in growth almost from day to day; it can disappear as rapidly, *e.g.*, after efficient surgical drainage of an interlobar empyema. Naturally this form can slowly replace the more usual tuberculous type in long-standing fibroid cases, where bronchiolectasis, if not true advanced bronchiectasis, is a common and expected complication. The patient in this case is suffering more from the consequences of his disease than from present activity, and bronchitis or bronchiectasis is all too easily given

as a label to one who is a chronic source of tuberculous infection to all his contacts.

In this connection we may note that emphysema is given as one of the causes of clubbing of the fingers. From our considerations of pathology we shall see that emphysema is almost always a secondary condition. Its *primary* cause is the *reason for* the finger changes in the vast majority of cases.

(*b*) Inspection of the chest as a whole should be made before any palpation methods are employed. Much confusion can result in all examinations up to film inspection if the presence of the "normal abnormality" of scoliosis is not recognised from the beginning. A general inspection of the chest, back and front, should warn the examiner of the true condition, and keep him from making some diagnosis of pathological import. It will prepare him also for those changes from the absolute normal which he will see on the radiograph, in the shape and position of the heart, and in the prominence of hilar and large pulmonary vessel shadows.

PALPATION. (*a*) The Sterno-mastoid Sign (see Figs. 1 and 35).

Several methods of diagnosing mediastinal shift by assessing the displacement of the trachea have been described. Most of these rely on deep palpation in the supra-sternal fossa, a procedure to which most patients object very strongly. A much easier and more reliable method is that to which reference is made several times in the course of these studies. It consists in gentle palpation across the tendinous origins of the sterno-mastoid muscles at the upper anterior surface of the manubrium.

We have seen in various chest conditions how the apex beat is not nearly so reliable a guide to mediastinal displacement, how the heart swings on its long axis much more readily than it does bodily to one or other side, and how for this reason, while it responds to inspiratory movements as shown in screening of cases of atelectasis, it does not give at positions of rest the same amount of fixed change to act as a guide to the examiner. Thus it moves backwards (from left lower lobe emphysema) in right lower lobe collapse, and its apex beat to palpation or stethoscope gives no aid to the actual diagnosis, while the sterno-mastoid sign will be strongly positive on the right side.

The main value of the sign is seen in cases of unilateral disease:

Fig. 33. Positive left sternocostal sign.

[To face p. 43.

it is only to be expected that bilateral disease will give bilateral changes of similar type. The onset of disease in the left side, following an established disease in the right, will inevitably undo the value of the sign, previously present in the right sterno-mastoid.

In acute conditions such as uncomplicated pneumonia, broncho-pneumonia and lung abscess, as in "cystic disease of the bronchi" and the epituberculosis of childhood, it is negative. We have discussed the reason for this. These diseases do not destroy elastic tissue, and have no effect on the normal pressure as usually exerted by residual air in the alveoli. In chronic conditions with loss of elastic tissue, atelectasis, and later with fibrosis, it is positive, *e.g.*, in tuberculosis, and bronchiectasis.

For its discovery the patient should be placed in a good light, standing, sitting or lying in a relaxed position, with the head central to thorax. Gentle palpation with the index finger across the muscle tendon is quite sufficient; the sharper edge of the affected tendon, which as against its softer, rounder neighbour will resist pressure, is easily felt. With very little practice the observer will see the sign before he applies palpation.

(*b*) Lung Movement

Many students attempt to assess lung movement by placing palms of the hands over the mammary regions with the fingers pointing upwards and outwards towards the outer subscapular regions. By so doing they cannot hope to find anything beyond the anterior, forward movement of the thorax, which is minimal as against the downward excursion of the lungs in response to diaphragmatic contraction, and the lateral movement in response to the increased sub-atmospheric pressure of the intra-pleural spaces. The assessment of downward movement by percussion will be discussed below. For proper assessment of lateral movement by palpation, the palms and closely approximated fingers should be placed gently but firmly in contact with the axillæ, with as wide an angle as possible between the fore-fingers and thumbs. The whole hands are then moved inwards, still in close contact with the chest walls, until the thumb-tips meet in the mid line of the sternum. The patient is then asked to take in a long, slow breath, filling his chest without heaving up his shoulders. Lack of movement on one side as against the other is immediately evident; it will not move the palm

fingers outwards, and the thumb on which these act as a lever will move but little from the mid-point as against its fellow. If the method is properly employed expiration should bring the thumb-tips together again at the mid-point of the sternum. The method is much more easy of application posteriorly.

Where bilateral emphysema is present the thumbs move from the mid line hardly at all, while the whole hands move upwards rather than outwards with deep inspiration.

PERCUSSION

(*a*) General. The note in the lungs is individual and comparative one side with the other. The normal for any patient should be assessed on the finding at the left apex to gentle percussion, with the understanding that while sound enters into such assessment the feeling of resistance transmitted to the plexímeter finger by the underlying tissue is of equal importance. Heavy tapping produces much noise but little to add to the knowledge of the examiner. Grades of departure from the normal can be described as impairment, dullness up to dead dullness, tympany and marked tympany.

A note on the left apex equal to that of the right must lead to a suspicion of left apical abnormality. Its pictorial representation by one line helps us to compare it with more marked abnormalities. Thus two lines will represent the usual dullness of tuberculosis and bronchiectasis, three the underlying resistance of pneumonia and fluid, and four the tonelessness and marked resistance of massive atelectasis and new growth. Tympany is found in the higher pitch and lessened resistance of the basal emphysema of chronic bronchitis, and marked tympany in the loss of lung tissue, replaced by air, in pneumothorax.

(*b*) Diaphragmatic Movement. Percussion to be complete should include examination of the change of note at the lung bases with inspiration and expiration, as by this means diaphragmatic excursion can be assessed. A normal diaphragm should move downwards at least one finger's breadth in the axilla, and one and a half breadths in the mid line of each half posteriorly, with deep inspiration. Lessening of extent of such movement will indicate adhesion up to complete fixture of the diaphragm. Examples of the usefulness of percussion in diagnosis are :—

(1) The dullness of upper lobe phthisis is predominantly

CHAPTER VII

AUSCULTATION

No student can hope to be able to distinguish the various abnormal sounds that can be found by auscultation unless he has accustomed himself to the many variations of the normal as they present themselves to his individual sense of hearing and tone. He should avail himself of every possible opportunity to examine healthy lungs in the fat, the thin and the muscular, using one uniform rule as his base line—*e.g.*, always listening in the upper axilla, or to the inner side of the lower inner border of the scapula, while the subject takes long quiet breaths through the mouth.

He will soon find the following three characteristics of the normal :—

(1) The sound is a gentle rustle, as if made by an infinite number of individual sounds that run together, an occasional one of these being slightly louder than its neighbours.

(2) Inspiration is slightly longer than expiration.

(3) Inspiration fades into expiration with no interval.

If now he will listen by placing his atethoscope over the trachea he will find four distinct changes.

(1) There is a distinct gap between the end of inspiration and the beginning of expiration.

(2) Inspiration and expiration are of equal length.

(3) There is an entirely new quality in the sounds. They are pitched higher than normal, as if the normal sounds of a large area were concentrated at one point, or the patient were blowing gently through a tube.

(4) The sounds are louder.

The first and second are present in every type of breath sound that is called "bronchial breathing"; the third and fourth vary considerably in different disease conditions, all of which are described as giving bronchial breathing.

Naturally every teacher demonstrates his own acuity of hearing and sense of tone. The writer has used the following rough rule of thumb :—

I. Over scattered areas of fibrosis and atelectasis, such as are found in chronic pulmonary tuberculosis and bronchiectasis, and

ance with their causative diseases, as these affect the bronchial and/or the parenchymatous structures. We hear the sounds known as sibili in both bronchitis and pulmonary tuberculosis, but whereas in the former we hear them mainly in the first or bronchial phase, in the latter they are mainly in the last or parenchymatous stage. Thus the same actual sound can be of very differing import to the examiner and the patient. We see why it is so easy for a case of active tuberculosis to be interpreted as merely bronchitis.

There appears to be considerable confusion in the minds of many students because they have no clear understanding of the true meaning of the many described abnormal sounds heard interfering with inspiration. They are so subdivided in the usual teaching that they lead to confusion. What is read by one physician as the only true sign of early tuberculosis may be read by another as evidence of a late stage of the disease.

For all practical purposes, the writer believes that added sounds can be reduced to three main types, each with the particular significance of its underlying cause. They are :—

(i) Crepitations, fine and coarse.
(ii) Sibili and rhonchi (coarser sibili).
(iii) Râles ; fine, medium and coarse (consonating or metallic).

1. Crepitations (see Figs. 37 and 38)

If the name crepitation be confined to a sound which has a dry quality to the ear, we can correlate it with fibrosis, *i.e.*, increased deposition of connective tissue, which we shall see occurs in various diseases considered under the section of applied pathology. The breath sounds are now so changed that their individual components appear to be a series of dry crackles. The coarser these sounds become, the less seem to be their number. They will indicate to us a permanent structural change which is not removed by cough ; a restricting material which lies round the bronchi and attempts to interfere with their natural expansion and elongation in response to the act of inspiration. In the pleural cavity fibrosis is the result of adhesion between pleural surfaces. We can hear crepitations, therefore, at two points in our examination.

(1) First, they can appear by the act of coughing. They are then fine, approximating to the sound produced by rubbing the hair above the ears. We are hearing the disturbance of interpleural adhesions which cough produces. They are a common

ing cavitation in each of them: it precedes the signs of cavitation and remains when they appear. As already stated, the crepitations are indicative of a change that will not recede, that may be masked by emphysema later, but may also lead to much more serious damage if the prime cause continues. If we read into them the lessons they try to convey, they will aid us in prognosis as well as in diagnosis; thus their continuance in the mid-phase, after the apparent complete evacuation of a lung abscess, warns us that our patient is not yet clinically well, that a so-called recurrence of his abscess may follow, and that what is really threatening is a consequent bronchiectasis, by involvement of the bronchus.

2. Sibili and Rhonchi (see Fig. 39)

These are best interpreted as indicating moisture obstructing the passage of air through bronchi, small and large. While very little moisture in the smaller bronchi will produce sibili, still smaller amounts in bronchi under contraction will give the same sound. The quality of the sound does not differ by the origin of the obstructing material: in other words, we can hear sibili that are due to stripping of the ciliated epithelium in acute bronchitis, and sibili that are due to material entering bronchi from breaking down parenchyma in phthisis. Again we hear them in acute asthma, because even the natural moisture, normally removed by the action of the cilia, cannot be dealt with; the last circular muscular fibres are contracting so severely in the terminal bronchioles.

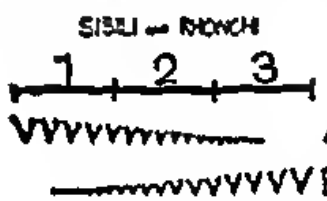

FIG. 39.
A. Bronchitis.
B. Pulmonary tuberculosis.

If, therefore, we apply our scheme of dividing inspiration into three parts we can correlate sibili and rhonchi with their pathological cause.

In the first phase they will be "bronchial" in origin: they indicate bronchitis and bronchiectasis.

In the second phase they will be "bronchial-parenchymatous." We are listening to diseases which affect both bronchi and lung, *e.g.*, broncho-pneumonia that is resolving, and lung abscess discharging. In the first they will be over a considerable area of the lung; in the second, they will be localised.

In the third phase they will be "parenchymatous" in origin:

They are still more insistent, and coarser, in the discharging lung abscess, although they are confined to one area as against those heard in the disseminated disease of broncho-pneumonia. They become more and more consonating and metallic where bronchial blockage has led to breaking down of the lung substance in a case now going on to chronic abscess. We hear now exactly the same sounds we heard in bronchiectasis. There we heard them begin and have their greatest intensity in the first or bronchial phase and they were heard over a fairly large area of the lung. Now we hear them in a localised area, and most intense in the mid phase of inspiration. The same pathological process is at work in both conditions. In bronchiectasis the disease progressed from the bronchus into the parenchyma: in lung abscess it progressed from the parenchyma to the bronchus. Chronic lung abscess is only a localised form of bronchiectasis.

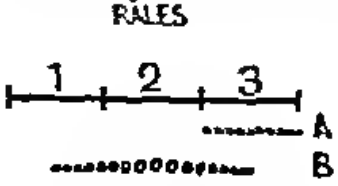

FIG. 40.
A. Pneumonia.
B. Bronchopneumonia.

In both cases, as the disease progresses, the rales get more and more metallic in type, telling us that communication between

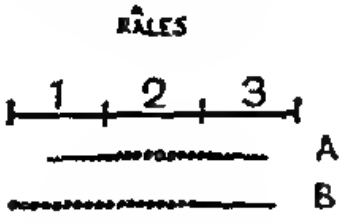

FIG. 41.
A. Lung abscess.
B. Bronchiectasis with lung abscess.

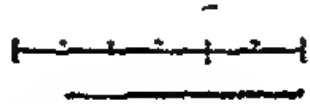
FIG. 42. Metallic râles in tuberculous cavitation.

cavitation in the lung and an enlarged bronchus is now established.

In the last, parenchymatous phase, we find them in pulmonary tubereulosis. With an advancing lesion they slowly replace the sibili and rhonchi of lesser obstruction; while, like them, they are due to material entering the terminal bronchioles, they are now due to actual breaking down of the parenchyma. As the condition goes on to estahlished cavitation the râles get more and more metallic, reaching their crescendo of sound at the very end of the inspiration.

The scheme of interpretation of added sounds is summed up in

SECTION III

CHAPTER VIII

SOME GENERAL POINTS, AND THE VISUAL, OBJECTIVE DESCRIPTION OF THE ABNORMAL FILM

In this section we shall gather together such changes from the normal as we shall see in the films of diseases discussed in detail in the section on applied pathology. It is possible to group them under their main characteristics. For example, most diseases of the lungs have in common an inflammatory exudate filling lobules as a result of irritation of their alveolar walls; this common process has a common homogeneous X-ray shadow. It is denser than that of thickened plastic pleurisy, but far less dense than that of atelectasis. It therefore follows that the shadows seen in these three separate conditions are all objectively described as homogeneous, although they may, by the experienced eye, be correlated with their individual underlying pathology, especially if they are studied in conjunction with the clinical details. These should always accompany the request for a report on the film of a patient who has not been clinically examined by the reader. In other words, a film report should be in two parts, the first a visual objective description of what is actually seen, the second an opinion on the cause.

It goes without saying that anyone attempting to give an opinion on an abnormal film must have an extensive knowledge of the normal, and of all "normal abnormalities." Reference has been made several times to difficulties that can arise by the reading of a pathological cause into the normal abnormalities of scoliosis, or simple calcification of the costal cartilages. Even the most experienced observer does not despise the assistance of a good example of the normal.

THE VISUAL DESCRIPTION

Some general rules for guidance may be set down :—

(1) Read every film according to a fixed routine, such as that given in the chapter on the normal film.

(2) In repeating on the trachea, heart and diaphragm, note any change in size, shape and position.

(3) In the lung zones look first for the vessel markings and the lesser fissure of the right lung, noting any increase, decrease or change in position, such as bunching or splaying of vascular shadows.

(4) Describe abnormal shadows according to their basic types, remembering that in front of them or behind them there may be shadows of normal structures. There are four basic types, streakiness, homogeneous shadows, non-homogeneous shadows, and ring shadows.

Streakiness is the name applied to increases in the normal lung markings.

Homogeneous shadows are those which do not alter in their evenness throughout their extent; although they may alter in density, they do not do so suddenly and interruptedly.

Non-homogeneous shadows are those that alter suddenly in density at one or more points throughout their extent.

Ring shadows have more or less circumscribing limits. In their centres they may have homogeneous or non-homogeneous shadows, or areas of transhceency. Similarly, their limiting boundaries may consist of one or other of these types of shadows. These shadows will be described in more detail in the next chapter.

(5) Give the size and shape of individual shadows; they may be single or multiple; large or small; well or ill-defined, diffuse or along the lines of normal markings; confluent or patchy; round, oval or triangular.

The term "mottling" is often used in descriptions of shadows. It conveys the idea of a mixture of opaque and non-opaque areas. In this sense it can apply to such shadows as occur in the film of uncomplicated broncho-pneumonia. It is, however, often used to describe a mixture of homogeneous and non-homogeneous areas such as are found in the film of pulmonary tuberculosis, and some interpretations confine it to this meaning. It is therefore not a good word to use, as being in the literature half descriptive and half interpretative, and with no accepted definition under either heading.

CHAPTER IX

THE INTERPRETATION OF ABNORMAL SHADOWS. STREAKINESS, HOMOGENEOUS AND NON-HOMOGENEOUS SHADOWS

BEFORE giving an opinion on the diagnosis of a patient under review the examiner should now inspect the film more closely at about 1 foot from the viewing box, and then go back slowly to a distance of some 5 or 6 feet. The writer realises that this will not make the film stereoscopic; what it will do is to make individual, more opaque areas, within a shadow that looks more or less homogeneous on close inspection, stand out against any background common to them. Unfortunately, this point cannot be demonstrated by prints but only by the study of films on viewing boxes.

There will always be the difficult cases where shadows are so confused or ill-defined that their underlying pathology cannot be diagnosed. There is no such thing as the typical film for every disease; there are always individual differences. Furthermore, we all understand that X-ray examination is at best only an accessory to general and specialised physical and laboratory investigations. Consideration of the general condition, the temperature, the blood-count, cellular content of effusion, and the result of bronchoscopy and biopsy may have to come into the summation of the interpretation, together with serial film examination, before we know in any one hypothetical case whether we are looking at a lobar atelectasis which is basically post-pneumonic, post-operative or due to carcinoma of the bronchus. There are, however, from the experience of various disease conditions as reviewed under applied pathology, certain guidances to be had if we look now more closely into the main types of shadow defined in the last chapter. It is understood, of course, that, while these are read individually, their accompanying shadows, and the evidence of interference or non-interference with other structures of the thorax, are read with them for interpretation. Thus, the shadows of pneumonia and of atelectasis of the right upper lobe are both homogeneous in type, but we have seen that there is no change in the blood supply of the middle and

lower lobes in pneumonia, whereas in atelectasis there is splaying of their blood supply, since they have become emphysematous.

Streakiness, and Absence of Normal Markings

Decrease and increase of normal lung markings are equally important. Decrease is found in pneumothorax, cystic disease of the bronchi, and emphysema. In pneumothorax the loss of markings appears outside the line of the collapsed lung, while this is offset by increased markings within it. With cystic disease some faint markings of normal lung in front of, or behind, the cyst may occasionally be seen, but usually there is a circumscribed area of the translucency of pneumothorax. Often there are with larger cysts definite increased markings outside the cyst, due to pressure collapse of surrounding lobules and the crowding together of their normal structural connective tissue and blood supply.

With emphysema there is a decrease over small or large areas of the lung, giving an increased translucency of the affected part. Where a considerable portion is affected, as in the gross basal emphysema of chronic bronchitis, or in the costo phrenic angle emphysema of the upper lobe due to lower lobe atelectasis, only the faint markings of the supporting structure of much distended lobules can be seen. Such a film diagnosis of emphysema may be of great import to the patient. As already stated, if it is not accompanied by apparent X-ray cause to ordinary film examination, it may call for special examination by lipiodol injection or bronchoscopy.

Increased streakiness is found in hyperæmia. We shall see how hyperæmia is the first indication of acute bronchitis, and the constant accompaniment, if not the basic cause, of chronic bronchitis. We have seen too that we ought to look for hyperæmic markings in a lung under collapse from pleurisy with effusion, and how we must suspect consolidation of the lung if we do not see them.

The distinctive feature of all shadows connoting fibrosis is that they stand out in increasing relief if we observe the film as we go backward to some 3 feet from it. This characteristic they share with those shadows we have named non-homogeneous. In chronic bronchitis they are almost invariably above delicate lines, as indicating that there is increase in peribronchial connective tissue caused by the hyperæmia of its vessels. With

fibrosis we may see corroborative evidence of the true pathology in disturbance of the normal position of other recognisable structures, *e.g.*, change in the position, or density, or both, in the lesser fissure, rise of the diaphragm, and perhaps displacement of of the heart and mediastinum in such a disease as bronchiectasis. Again we see streakiness, perhaps dissociated from the usual normal markings, in carcinoma, especially in the nodular-medial and basal types. We find it also, with or without discrete nodules, in the reticulations of silicosis, when we are viewing the congested and thickened lymphatics associated with this disease.

Homogeneous Shadows

These shadows, when in the lung, result from exudates and de-aeration ; in the pleural cavity they are due to exudates and occasionally to transudates.

On the whole it can be taken as a safe rule that homogeneous shadows indicate acute lung disease. They are found in pneumonia, broncho-pneumonia and in the acute, progressive parts of tuberculous lesions. Their variation in density can give us a hint of their underlying pathology. Homogeneous loss of translucency of lesser density is found as the X-ray evidence of inflammatory reaction in the alveoli. This is due to irritation of the walls of the cells by the organisms which later penetrate the walls and fill the cells with exudate. Such exudate throws a denser but still homogeneous shadow.

It follows that under this type of shadow falls that called by the name of "pneumonitis." This term is justifiable if it is merely descriptive and not meant to be specific and interpretative. It is not a pathological entity. It is the shadow common to every process irritative to lung parenchyma, being the œdema or catarrhal exudate seen in pneumonia, broncho-pneumonia, lung abscess, bronchiectasis and tuberculosis. Its presence means that some specific disease is at work ; we shall by serial films find this underlying cause, or we may require lateral or tomograph films or even such investigations as injection of lipiodol or bronchoscopy to reveal it.

In uncomplicated pneumonia and broncho-pneumonia we see a homogeneous shadow involve the broncho-pulmonary areas of a main bronchus or one or more of its branches. In phthisis we see it in the localised areas of dorsal bronchioles. In both we can follow its progress from the catarrhal to the specific heavier

which shows that these materials are replacing the residual air, and not altering the normal pressure which residual air maintains.

Again, in the film of adult proliferative phthisis we have, at some stages of the disease when exacerbations are taking place, the possibility of three homogeneous shadows. First there is a generalised, fine loss of translucency forming a background to all other shadows; this is the plastic pleurisy of slow irritation and adhesion of the overlying layers of the pleura. Second we see scattered areas on the edges of the main shadows; they have a density slightly greater than the first, and equal to that of the catarrhal exudate of broncho-pneumonia. Next we see the denser homogeneous shadow of tuberculous exudate in lobules where the tubercle bacilli have invaded bunches of acini to produce true tuberculous exudate. None of these alter materially on distant viewing, while all three incline to merge when we look very closely. The distant view will, however, throw into strong relief any non-homogeneous shadows also present.

Non-homogeneous Shadows

These are shadows which alter materially in their density within their extent on viewing at ordinary distances, and still more so when viwed at a greater distance. As against homogeneous shadows, which mean acute lung disease, we can take it as a safe rule that they mean chronic disease—bronchiectasis and pulmonary tuberculosis being the commonest. In other words, we can correlate them with fibrosis and organisation, and with the type of atelectasis which results from these two processes They are denser, more clear cut and harder *looking*, and *usually* very irregular in outline. They may on close inspection appear to be confluent with the rest of the abnormal shadow of which they are part, but on distant viewing they stand out sharply. This is because they are surrounded by lobular complementary emphysema, the continual accompaniment of atelectasis in all its forms. We have already noted that there can be no collapse, in either small or large areas of the lung, without corresponding emphysema. The translucency of these emphysematous lobules acts as a foil; it offsets the organised areas. It is therefore possible to write an interpretative report of "tuberculous infiltration, fibrosis and pleural thickening."

affections up to the sepsis of bronchiectasis, and can sometimes be recognised in a collapsed lobe. Ordinarily, however, in their uncomplicated state they have two definite characteristics in addition to those stated which can aid us to give an interpretative reading. First they have no associated evidence of fibrosis round them; second they have no associated mediastinal shift.

The outline of lung abscess which is discharging gives the next more evident homogeneous ring shadow enclosing an area of comparative translucency. The boundary is now round $\frac{1}{8}$ to $\frac{1}{4}$ inch in depth. It usually has a generalised hazy loss of translucency external to it and over its enclosed lung tissue, this being due to thickened pleura, and, on its outer edge, to catarrhal exudate in those alveoli which have been irritated but not actually involved in the disease process. The actual boundary is made of pneumonic exudate and so does not alter materially in whole or in part with distant viewing as against closer inspection. It has no surrounding fibrosis. It is therefore in every way a single entity in the lung field, oval rather than rounded. Its homogeneous character tends to remain up to its change to the secondary stage of bronchiectasis, its inner pneumonic constituent appearing to be frozen *in situ*, even while its outer lining shows increasing and decreasing densities with advancing invasion of its sepsis into the surrounding lung. Moreover, it still remains an entity, and not just part of a generalised involvement of the particular lobe or of the lung field in which it is situated, as is nearly always the case with the tuberculous cavity. Apart from the acute excavation of an Assmann's focus, a tuberculous cavity is seldom without definite surrounding fibrosis.

The average tuberculous cavity can be visualised from what has been already described (see Fig. 90, p. 111). Its surround in both internal and external borders is irregular and uneven. It merges in its outer zone with evidence of fibrosis. This fibrosis, with organised and collapsed lobules that are fused together and opaque, form its wall, which stands out in bold relief to distant viewing. It is seldom the only evidence of disease in the lung. It may contain a fluid level, which shows as a homogeneous shadow with a straight, horizontal upper border, but a fluid level is much more common in the lung abscess which is not evacuating freely because the bronchus connected with it is blocked.

(*b*) **Ring Shadows round Areas of Comparative Density.** Shadows may be of ring shapes round areas of comparative loss

FIG. 43 Assmann's focus in right upper lobe.

[To face p. 61.

SECTION IV. APPLIED PATHOLOGY

CHAPTER XI

BRONCHITIS AND EMPHYSEMA

Acute Bronchitis

In bronchitis both acute and chronic there are two changes from the normal of which we must take particular note. These are increased blood supply, and more or less destruction of the inner constituents of the bronchial wall. The increased blood supply contributes to our film findings. We have noted several times that the branches of the bronchial tree are closely associated with those of the blood vessels and that the latter show on a normal film. In bronchitis these markings become more evident, being due at first to the congested blood vessels, and later to increased connective tissue round the bronchi as a result of this hyperæmia.

In acute bronchitis congestion causes swelling of the mucosal coat, while all the coats become infiltrated with large numbers of polymorphs. As a result the ciliated epithelium peels off, and, mixed with degenerated and broken up polymorphs, makes a stringy exudate on the mucosa to form the muco-pus which the patient expectorates. This naturally tends to obstruct the bronchial lumen, and, as air attempts to pass it, gives us the stethoscopic sounds of rhonchi and sibili in the larger and smaller branches. These sounds may be heard throughout inspiration, but if we listen critically we find they are most insistent in the first third of the breath sound and thereafter tail off towards its end.

This position in inspiration is an important diagnostic feature as against pulmonary tuberculosis. There is no fundamental difference in the quality of the sibili and rhonchi heard in both conditions, but the sounds denoting active tuberculosis do not appear until the third stage of inspiration and get more and more marked until its end (see Fig. 44).

If the condition is very acute, and so produces a heavy viscid exudation, this may be seen on the film in larger bronchi as a loss of translucency running between thin, ill-defined, parallel lines. The loss of translucency is due to the exudate on the

have been lost, so that there is much more obstruction by material which is now mainly pus from degenerated polymorphs. We see therefore why it is that the abnormal sounds of rhonchi are much coarser than those we heard in acute bronchitis, but also why we get, from less viscid sputum, less loss of translucency within the lines of more inflamed bronchial walls.

(*b*) It causes small round cells to invade all the bronchial coats from the mucosa outwards. These cells become infiltrated by capillaries, and the consequent deposition of fibroblasts, with endarteritis of the capillaries, leads to increasing fibrosis. The main brunt falls on the connective tissue coat, because of the long continued hyperæmia of its blood supply.

On the film we can recognise it in definite white streaks along the lines of the affected bronchi, continuing more clearly, and much further outwards from the hilum than any normal striations we may see connected with vascular markings or with the supporting structure of the lung. They are particularly evident along the inner bronchial bundles of the lower lobes on both sides; only the fact that the heart shadow covers less of the right lower zone makes them more apparent in this area as against the left lower zone. If we stand some distance from the film we can see these streaks give much more evident outlines to the affected bronchi.

Fibrosis gives us a new stethoscopic sign beyond the rhonchi of bronchial obstruction. We hear dry crepitations throughout inspiration, which remain after cough (see Fig. 45). These show us that fibrotic material round the bronchus is resisting its attempt to elongate and open up by the act of inspiration. What was previously only a supporting surround, expanding and contracting with the normal movements of the bronchi, is now a constricting band. As this material is static and not moveable, as mucus, by cough, it is a steady finding in all chronic inflammatory diseases of the chest. In its turn it produces emphysema, which we shall discuss a little later.

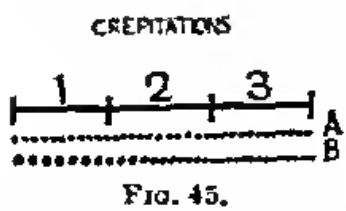

Fig. 45.

A. Generalised fibrosis.
B. Marked peribronchial fibrosis.

A still further effect is shown on the film (of the advanced case) in an alteration of the normal cardiac outline (see Fig. 31) due to resulting hypertension. An early sign of this complication appears to be an increasing size of the aortic knuckle, similar to

FIG 46. Segmental collapse of the right lower lobe in broncho pneumonia. The line of the collapsing lingula can be seen in the outer lower zone. Outside the defined edge of the affected segment there is emphysema in the costo phrenic angle.

[To face p. 65.

distension of lobules from shrinkage of their supporting connective tissue. We shall note later how this occurs in pneumonia which heals slowly and badly by repair instead of resolution; known as post-pneumonic fibrosis, the condition commonly affects the whole of one lung. The contracting peribronchial tissue drags on the septa of the lobules, bringing a generalised stretching of the elastic tissue of the individual alveoli, but it can also become so much thickened that it crushes and destroys the alveoli in its neighbourhood. In other words, we have both complementary and atrophic types.

All forms of emphysema occur most easily where the supporting tissue is normally weakest. This is why the lung bases so easily blow out in chronic bronchitis; why the lower anterior edge of the right lung so readily distends when the middle lobe collapses, to fill in the space vacated by the receding lobe. It is the reason too why, with the collapse of one lower lobe, the other one so readily bulges across the anterior mediastinum in an attempt to take its place. The lack of support is the cause why we occasionally see localised emphysema over the apices of the lung, that may rupture without obvious cause of strain and present us with "simple spontaneous pneumothorax" through leakage of air into the pleural cavity (see Fig. 11, p. 6). It is this same natural weakness that will cause the outer and upper apices to suffer first in blockage of the upper lobe bronchus; their lobules collapse very easily and so lose their residual air. Thus it is sometimes possible to see a slight loss of translucency, of even, homogeneous type, in the outer infra-clavicular region quite early in a case of carcinoma of the upper lobe bronchus.

We have already learnt how elastic tissue allows expansion of the lung, as it responds to muscular stretching throughout the bronchial tree, and how it finally confines the extent of such expansion by its network terminations round the alveoli. Neither action takes place when emphysema is present. Affected parts have lost their power of movement, as they are constantly at full distension. They can be moved only bodily, being dragged by other unaffected lung structures. Hence the heaving, non-lateral movement of the lung bases in chronic bronchitis, and the permanent hyper-resonance from constant expansion, which has a tympany definitely beyond that of forced inspiration in normal tissue. Prolonged expiratory effort is to be expected from the sufferer; nature is attempting to force air out of the blown-out

vesicles. We see why expiration is now equal in length to, or longer than, inspiration.

Blood vessels in the alveoli and in the supporting connective tissue of lobules are drawn out into thin wiry structures, and some are occluded altogether, with two results. First, there is a vicious circle; the septa cannot live without blood supply and so break still further; second, the narrowed vessels in the alveolar walls resist the pulmonary circulation and so reflect on the right heart and open the way to all the stages up to congestive failure. Close inspection of the film shows a corresponding change in the pulmonary arteries, which are swollen, and evident much further out from the hilum than in the normal film.

The reason is not far to seek. Such dragging out and occasional obliteration of the capillaries in the alveolar walls means that less blood is open to the residual air: there is considerable lessening of gaseous interchange; less chance for the liberation of carbon dioxide and its replacement with oxygen. At the same time there is less movement of the alveoli; they cannot get the steady renewal of residual air. This is the explanation of the cyanosis of the chronic bronchitic, and also of the increasing cyanosis in the "bronchial asthmatic." He has the added difficulty of the muscular contraction that brings closure of the terminal lumen to the acinus. We saw the reason in discussing the muscle fibre structure of the bronchial tree: it ends in circular fashion round the last recognisable lumen at the entrance to the acinus (see Fig. 18, p. 10).

The alterations on the X-ray film of an advanced case are now easily visualised. The rib inter-spaces are widened. The vesicles are fewer and larger than normal, so there is an increase of translucency by decrease of striations due to their walls. The pulmonary vessel markings are more in evidence. As there is less venous return the heart is smaller. The diaphragm is flattened as the blown-out vesicles press against it. We can see the cardiophrenic angles opened out, and to percussion we find much decrease in the muscle movement downwards with inspiration. The continual flattening slowly brings paresis of the muscle, with consequent basal stasis of the accumulated *débris*.

CHAPTER XII

BRONCHIECTASIS

Bronchiectasis means a widening of the bronchial tubes. This may occur over a length of the bronchus in the form called cylindrical, or be localised in the form called saccular. It may be "congenital" in the sense that it is a complication of a prior developmental abnormality of the bronchi, but is usually recognised as being frankly an acquired condition. For its production both obstruction of the lumen and infection of the weakened walls are necessary. Because of the liability of *débris* to accumulate by gravity and stasis in the dependent parts of the lungs we find that most cases are basal, but it may appear in any part of the parenchyma as the result of blockage of its supplying bronchial lumen. Where small branchings are thus involved we have bronchiolectasis. We shall see that this is a continual accompaniment of the grosser form involving the main branches of the lower lobes.

The pathogenesis is most easily understood if it be envisaged as a further development of chronic bronchitis, which has become ulcerative. After weakening the wall by its chronicity it has destroyed all the components of the wall from the epithelium to the muscle and elastic fibres, and has finally penetrated the cartilage to infect the surrounding parenchyma.

By the loss of its muscle the wall can no longer maintain the resting-period peristalsis necessary for the renewal of residual air to its dependent lobules; by the loss of its elasticity it can no longer elongate with inspiration and recover with expiration. The destroyed material of the components in the wall interferes with the natural movements of the cilia. The marked increase in blood supply brings the same repair effects as we found in chronic bronchitis, but with much more exaggeration; gross cellular infiltration of the walls and the surrounding connective tissue leads to considerable fibrosis. This fibrosis by its constriction interferes with the movement of the bronchi, and by its contraction pulls on, and therefore widens, the weakened walls. The obliteration of the blood supply derived from the bronchial arteries adds to the vicious circle; they close down by endarteritis.

The effect on the lobules dependent on the affected bronchi is disastrous. From the first their supply of residual air is lessened. Later their supporting septa become involved in the advancing fibrosis, and when their supplying bronchioles become occluded by the septic material they either collapse if empty, or organise if already filled up by exudate. In the chapter on the mediastinum we have seen how this reacts on the surrounding lobules to produce complementary emphysema, and on the bronchioles to produce cavitation; in other words, we have bronchiolectasis around bronchiectasis.

The physical findings therefore begin as a coarse chronic bronchitis. Rhonchi are found in both inspiration and expiration, because the cilia have gone, and we are left with cubical epithelium. Fibrosis brings a change in breath sounds. The pitch is higher, and we notice a gap between the end of inspiration and the beginning of expiration over a considerable area of lung. The crepitations of fibrosis take on a particularly insistent character and become more and more marked in the first third of inspiration. The lung movement is heaving because of the increasing basal emphysema which gives us also an overlying note of tympany to percussion. We find however that there is an underlying note of dullness, and a resistance to the pleximeter finger due to the fibrosis and the organisation of the parenchyma round it. Because the mediastinum is dragged and pushed to the diseased side we get a positive sternomastoid sign on the same side, if the disease is unilateral.

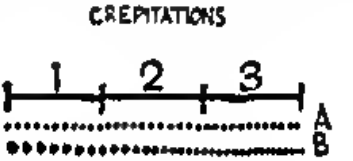

FIG. 47.

A. Generalised fibrosis.
B. Marked peribronchial fibrosis.

The X-ray film can be understood and correlated with these findings (see Fig. 20, p. 13, and Fig. 48). There are heavy striations towards both bases, due to the increased blood supply and then later to the increased connective tissue. At first therefore these striations are more or less faithful to the normal branchings of the pulmonary artery, but as the fibrosis begins to tell there is shrinkage of the bronchi into the cardio-phrenic angle. We can see them bending inwards, instead of going outwards and downwards. As the opacity increases in the cardio-phrenic angle translucency increases in the costo-phrenic angle, as this area, vacated by the shrinking lower lobe, is being filled in by the emphysematous upper lobe. Lipiodol injected at this stage will

show how destruction of the wall is by now involving the cartilage, to produce ill-defined sacculations (see Fig. 49).

The film of early bronchiectasis, with crowding of the bronchi into the cardio-phrenic angle (see Fig. 20, p. 13), is rather like that of a descending upper sinus infection. This is a very common finding, often accompanying an acute antrum trouble. Expert opinion on the upper sinuses, including X-ray, should, of course, be obtained, but the following points may aid in comparing the chest films. The film due to upper sinusitis usually shows much more homogeneity in its basal shadows; we do not get the same evidence of fibrosis unless the condition is of very long standing, with continued reinfection of the bronchi, when, of course, the patient is developing resultant bronchiectasis. Further, the film shows a confused shadow of the inner third of the diaphragm merging into that in the cardio-phrenic angle. We do not see the lower bronchi curving inwards. We are looking at hyperæmia of vessels continuing below the diaphragm.

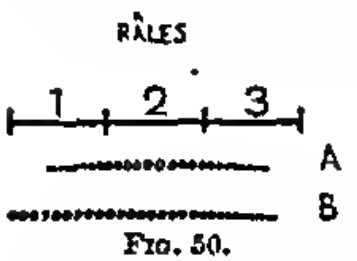

FIG. 50.
A. Lung abscess.
B. Bronchiectasis with lung abscess.

If we look at the film of bronchiectasis at some distance from the viewing box we shall see that the striations stand out clearly as well-defined parallel lines, here and there interrupted where the cartilage is disappearing. Around them we see small opaque and hard-looking shadows, which are due to the collapsed and organised lobules, well defined because they are offset by others among them which have complementary emphysema. That is, the disease is extending by bronchiolectasis.

The next stage is perforation of the bronchial wall and consequent extension of the sepsis into recognisable areas of parenchyma. Its occurrence is heralded by the stethoscope finding of râles, which mean that parenchymatous material is entering the bronchi (Fig. 50). If we listen critically we shall find they are in the middle third of the inspiration, that is, the "bronchial-parenchymatous" phase. They rise in intensity to it from the first phase and then tail off in the third. They are due to air entering bronchioles into which catarrhal exudate is discharging from lobules that have been filled with that first inflammatory fluid, which is the indication of irritation of their walls. In other words they are the râles of localised lung abscess.

We can recognise this exudate on the film. It is the "œdema"

FIG. 48. Bilateral basal bronchiectasis

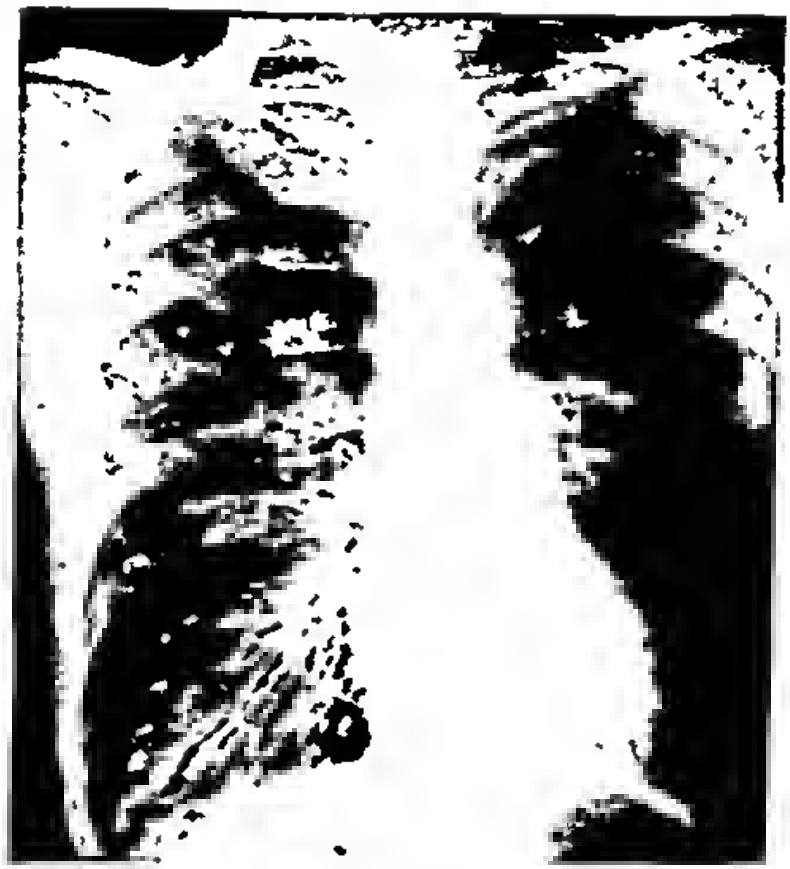

FIG. 49. Lipiodol in basal bronchiectasis.

of many radiological reports, and is always homogeneous in its shadow, having a light loss of translucency comparable with that thrown by the inferior vena cava. In this particular instance it is seen on the outer edges of a more opaque and likewise homogeneous shadow, cast by lobules that have passed the stage of inflammatory exudation and are now filled with septic material. We know it connotes advancing bronchiectasis only because we read with it the characteristic shadows of fibrosis and organisation detailed above. It appears with each exacerbation of the disease, later gets denser as becoming true septic exudate, and later still shows all the non-homogeneous changes of collapse and fibrosis.

What we have now is a cavity in the bronchus connecting with a cavity in the lung: septic bronchi are continuous with septic parenchyma. To all intents and purposes we have in several areas what we shall find later is the end-result of a chronic lung abscess in one area, where the septic lung has infected the bronchus supplying the diseased broncho-pulmonary segment. We shall find that the râles have taken on a distinctive metallic, consonating quality. These sounds are now present throughout two-thirds of the inspiration, over a considerable area of the lung field.

Lipiodol introduced at this stage will demonstrate every step of the destructive process; here we shall see only cylindrical enlargement: there sacculation; at still another point nothing resembling a bronchus, but only a large irregular opaque area with cotton-wool edges, which merge with the finer loss of translucency that indicates catarrh in irritated lobules.

Mediastinal shift will be more evident still to both film and physical findings. The fingers will show gross clubbing of the drum-stick, septic type. Dullness will be much more marked, and the foul sputum will contain the elastic fibres of the destroyed parenchyma.

Bronchiolectasis

We have noted how bronchiectasis multiplies itself in the minor form of bronchiolectasis by the involvement of bronchioles. Such a condition is almost always a secondary one; the lumen has been blocked, and then infected, by septic material in the lobules it supplies, a process which copies the pathogenesis of adult tuberculosis. We shall see in the next chapter that its common cause is primary broncho-pneumonia, which has not been able to get rid of its exudate. It is always a fairly localised condition, and this helps us to differentiate it as a picture that has

elements not dissimilar to those of broncho-pneumonic tuberculosis, where the process is much more widespread into the mid-zones.

It is not the same condition pathologically or radiologically as so-called "bronchiolitis obliterans." This condition is the end-result found with Friedlander's bacilli and the bacilli of influenza, which are carried along the bronchi to the terminal bronchioles, and thence direct to the lobules they supply. These organisms seem liable to attack the parenchyma directly, not reaching it by way of the connective tissue as the pneumococci of pneumonia or the streptococci of broncho-pneumonia, as we shall see in the next chapter. When their exudate organises in the lobules we get scattered areas, in clumps of tiny dense opacities all over large areas of one lung, and more usually of both lungs. They are really "scattered lobular obliteration" or "disseminated focal pneumonia," and they lead to as widespread complementary emphysema. Bronchiolectasis is a very uncommon result. The X-ray is similar to that seen in many cases of blast injury of the lungs.

Since the introduction of mass-radiography the author has seen quite a number of films of this type. Usually the patient is quite symptomless; he may or may not give a history of a recent cold. Serial films have shown that many clear up completely over a shorter or longer period.

The picture cannot be differentiated from that seen in patients who have inhaled paraffin particles from long continued nasal medication, or from that seen in several cases of shipwreck survivors who have inhaled oil.

CHAPTER XIII

THE PNEUMONIAS

THE pathogeneses of pneumonia and broncho-pneumonia are fundamentally the same, consisting in the interstitial reaction to the infecting organisms. In both diseases the organisms penetrate the wall of the bronchus to its surrounding connective tissue, and by its lymphatics are carried to the walls of the alveoli, which they first irritate, and later penetrate. In this way the pneumococci of pneumonia and the streptococci of broncho-pneumonia differ from the bacilli of influenza and Friedlander's bacilli, which we have just noted are carried direct to the lobules by their supplying terminal bronchioles.

The connective tissue puts up a poor resistance to the pneumococcus. In the great majority of cases the organism seems to penetrate the wall of a main bronchus, such as that to the right lower lobe, close to its origin. It finds no protective barrier against its invasion of the parenchyma and therefore rapidly involves the whole lobe. On the other hand, the connective tissue puts up an intense resistance, shown in marked inflammatory reaction, to the streptococcus, so that it confines the disease to patches of lobules. We can see why lobar pneumonia is a disease which spreads from the hilum in radial fashion. Its progress is rapid: the invading army is too strong and sweeps aside any slight opposition it may meet. It is possible, however, now and again to see an example where pneumonic exudate is already developed near the hilum before the first evidence of inflammatory reaction is evident in lobules nearer the periphery.

The manner of onset explains to us also the manner of clearance by either resolution or repair, which must be again radial from the hilum to the periphery. The bronchi have a chance to recover from any slight reaction in their connective tissue before they are called on to deal with the parenchymal exudate, which will flood them in due course to produce the typical pneumonic sputum. This manner of clearance of the disease is an important one in differential diagnosis by film examination. It is the opposite to that seen in the so-called "epituberculosis" of childhood. Serial films will help us to decide which we are

dealing with. The picture of pneumonia clears rapidly after the crisis, but epituberculosis will take many weeks, even months, to resolve, and will leave on the film enlarged and crenated hilar glands and perhaps also a well-marked "Ghon's focus" at the site of the original infection towards the periphery of the affected part.

Let us try to follow the pathogenesis of a developing pneumonia in more detail as we see it on the film. If there is any reaction in the connective tissue to the penetration of the bronchial wall by the pneumococcus we see increased striations along the lines of bronchial branching due to congestion by polymorphs in the peribronchial, perivascular and inter-lobular connective tissues. These may be evident in the outer zone of the affected lobe while its inner area gives a homogeneous loss of translucency. This shadow is of the same type as we found in the extension of sepsis from bronchus to parenchyma in advancing bronchiectasis. On the outer edge we see the same finer loss of translucency due to the non-specific irritation of alveolar walls. Nearer the hilum we have the denser shadows of true pneumonic exudate, consisting of polymorphs, some large mononuclears and lymphocytes, in a serum containing a varying quantity of fibrin dependent on the connective tissue reaction. The lobules so affected fill quickly and distend, and this stretching of their elastic tissue helps them to contract later so that they can evacuate the exudate into their bronchioles.

It is fundamental to the understanding of the physiological effects, the X-ray film and the physical signs, to realise that when pneumonia is uncomplicated this exudate replaces the residual air in every sense. The same physiological action as that of residual air remains on the elastic tissue of the bronchi. The alveoli are filled to full capacity and the elastic network of the alveoli is therefore on stretch just as it would be in full inspiration. There is no blockage of terminal bronchioles, and therefore no collapse of lobules, and no resultant shift of the mediastinum to the side of the disease. The same thing applies to uncomplicated broncho-pneumonia and uncomplicated lung abscess, so that they are totally unlike in pathogenesis, physiological effects and physical signs, to both bronchiectasis and adult phthisis. For these latter are proliferative diseases. They cause organisation and collapse of lobules, and have from their beginning associated fibrosis. They will therefore cause the mediastinum

to drift towards the part of the lung they affect, so that we shall find the heart and the trachea displaced towards them.

Once fully developed, pneumonia outlines the lobe anatomically, and the dense homogeneous shadow of its exudate remains until the crisis. This being so, the shape and density in any one lobe must vary with the shape, and alter with the depth of tissue in the normal lobe, as we saw in our reminders on applied anatomy. The right middle lobe must be defined and dense just below the line of the lesser fissure, and become more translucent towards the lower zone, and unless the lower lobe is also involved we must see a clear area between the lower limits of loss of translucency and the diaphragm, because this area is occupied by the lower lobe (see Fig. 22, p. 18).

In the same way the shadow of lower lobe pneumonia, densest in the lower zone over the diaphragm, must lessen steadily upwards towards the mid zone. It is sometimes confused with that of tuberculous pleural effusion. The physiological effects to be expected in these conditions should aid us; effusion pushes the mediastinum to the opposite side, where we shall find a positive sterno-mastoid sign. If we look critically at the shadow of tuberculous pleural effusion we shall see that its peripheral density is far less than that of pneumonia, and that we can usually see the ribs through it (see Figs. 5 and 93, pp. 4 and 110). Its loss of translucency increases steadily towards the middle of the lung field, giving an impression of a band running from the axilla downwards and inwards to the diaphragm. The effusion is compressing the lung, and we are looking at the area so compressed when we see this band of greater density. This is quite unlike the pneumonic density, which is equal at any one level throughout its extent along a horizontal line drawn between the periphery and the mediastinum. Here the density must lessen as it goes upwards, as the lobe thins out to its lingula; in other words, there is less and less depth of parenchyma to be filled by the pneumonic exudate.

Moreover, we shall remember the further aid to differential diagnosis. An uncomplicated pleural effusion will not blot out the vessel markings in the underlying lung but exaggerate them, as there is hyperæmia, but pneumonia blots out all normal striations and is usually so dense in its shadow that we cannot make out the outlines of the ribs.

The picture of fluid complicating lower lobe pneumonia can now be readily visualised, for it combines both pictures. Towards

the periphery we shall see the lighter loss of translucency of the fluid, and through it we can generally see the ribs quite clearly as far as the outer edge of the denser shadow cast by the pneumonia. No lung markings will appear in either shadow. Now the mediastinum will be to the opposite side, whereas it was central as long as the primary pneumonia was uncomplicated. Before the fluid developed there was no sterno-mastoid sign: now the muscle on the opposite side is under distinct tension.

From what we have learnt so far it follows that the slight and temporary shift, sufficient to be noted by film examination in some cases of pneumonia, is due either to overdistension of the lobules by their exudate or to an ephemeral blockage of their bronchioles. Close inspection of the picture should tell us which we are viewing, for the effect of the former will be traction on the mediastinum without evidence of collapse; the latter will present accompanying evidence of what has taken place. For example, if this has occurred as a complication of right upper lobe pneumonia we shall see a distinct lower edge of the density, in a sharp line. This is proof that the lingula of the lobe has shrunk upwards to the line of the lesser fissure, which becomes convex upwards, and is higher than its thin hair-like streak we can see about the third or fourth interspace on the normal film. Furthermore, the uncomplicated pneumonia showed normal blood vessel markings in the middle and lower lobes, but now these are splayed out in order to supply the emphysematous mid and lower lobes.

To physical signs pneumonia gives us dullness due to exudate. As the lobular elastic tissue is on full stretch and maintains the inspiratory position of the bronchial walls, we have no movement of the affected part. There will therefore be absence of true breath sounds, but bronchial breathing will occur, as the air entering the main lobar bronchus at its origin is conducted through the consolidation which acts as a sounding board.

The method of clearance of the pneumonias and the rapidity of the process is a fortunate affair for the patient. Considering all the possibilities of complication, we may well marvel that we see so few cases of resulting fibrosis, for the peribronchial and perivascular tissues, so recently recovered from their initial reaction to the invading organisms, have now to deal with lymphocytes and plasma cells. These again produce striations on the film, and they can remain for a considerable time. They should not

lead us to give a bad prognosis, by reading them as evidence of fibrosis, unless we find they are accompanied by the characteristic crepitations of this condition. But they can produce fibrosis, as we shall discuss in more detail later.

The fibrinous exudate in the lobules is coughed up or absorbed after being liquefied by a proteolytic ferment supplied by its accompanying polymorphs. It is because of this liquefaction that we hear the so-called "redux crepitations" of pneumonia. If we listen to them critically we shall find that they are really sticky sounds at the very end of inspiration, and are râles rather than crepitations. We are hearing air entering the alveoli through the exudate which is now discharging into terminal bronchioles (see Fig. 51).

It may be that poor supply of polymorphs in the exudate is the basic cause in those cases that go on to fibrosis; capillaries have time to enter it before it is liquefied.

In any case we do know that fibroblasts replace the fibrin, passing through the alveolar walls and from lobule to lobule, causing organisation of the exudate that remains, and consequent shrinkage collapse. Although others around these lobules are bound to show some complementary emphysema there will be shift of the mediastinum to the affected side: normal unaffected tissue is being dragged over by the intra-pleural pressure. Chronic interstitial pneumonia, as it is called, is therefore exaggerated repair tissue. It is usually found in one lung, and we can see how it differs from the fibrosis of chronic bronchitis due to bilateral long-standing hyperæmia, because this latter is a peribronchial tissue increase, not an inter-lobular one. If it is very severe it can crush the supplying bronchioles and give us scattered areas of lobular collapse, which is not an unusual finding.

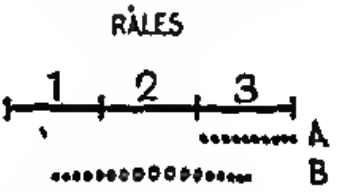

FIG. 51.

A. Pneumonia.
B. Broncho-pneumonia.

It follows that the X-ray of chronic interstitial pneumonia will show the ribs on the affected side closer together than normal, and that the diaphragm will be higher than normal, while the heart and mediastinum will have drifted towards the fibrosed lung. Throughout the diseased lung will be strands of fibrosis, which have no distribution corresponding with that of the normal blood supply. These will be more prominent on distant viewing,

and will be seen to have scattered among them nodular-like shadows, which are cast by organised bunches of alveoli.

Auscultation will give us widespread crepitations which reach their loudest point towards the end of inspiration, as they are parenchymatous in origin.

This condition is quite unlike the results of the "pneumonia of the new-born," because here atelectasis is a preceding entity; the lobules never opened at all, which is necessarily a different condition from acquired collapse. It is also quite unlike so-called "post-operative pneumonia," which is not pneumonia, but a purely bronchial disease. A sudden thick, sticky exudate from the wall blocks the lumen, with the same dramatic effect seen with an inhaled foreign body. Residual air cannot be renewed; the parenchyma collapses, and there is no obstacle to the action of the intra-pleural pressure on a lobe that perforce must shrink to its main bronchus at the hilum. Screening the patient or taking two films, one on complete inspiration and the other on complete expiration, will show us what never happens in ordinary pneumococcal pneumonia: a swing of the collapsed lobe, the heart, and the mediastinum, with each inspiration, to the affected side. There will be some recovery with each expiration, but still a displaced mediastinum, which will be shown by the positive sterno-mastoid sign even if it be impossible to demonstrate by the position of the apex beat. If the glutinous material is not cleared out by postural drainage or bronchoscopic suction the consequences can be very serious. Organisation can lead to every stage of bronchiectasis, the same end-result so often seen in the "pneumonia of the new-born." To this last condition the name "congenital bronchiectasis" may be applied; with its atelectasis, present from birth, there is often mal-development of bronchi, so that cysts are frequently found, and these the superimposed infection turns into sumps of septic material to add to the bronchiectatic end-result.

From what has been said of the pathogenesis of pneumonia the possibility of localised forms can be easily envisaged. The organisms travel some way along the main bronchus before piercing the wall of one branch. This is comparatively common in the pneumonia of childhood, which gives in one of its forms the so-called "hilar pneumonia," by involving the posterior horizontal branch of the right lower lobe bronchus. It is not uncommon to find in an adult that only the pectoral or the axillary branch of

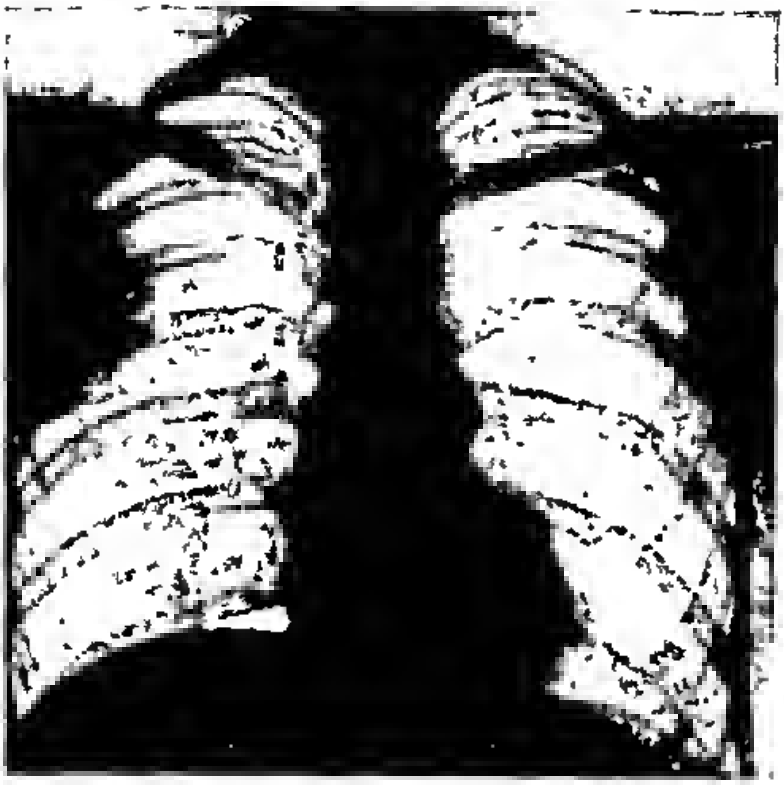

FIG. 52. Pneumonia of the axillary branch of the right upper lobe bronchus

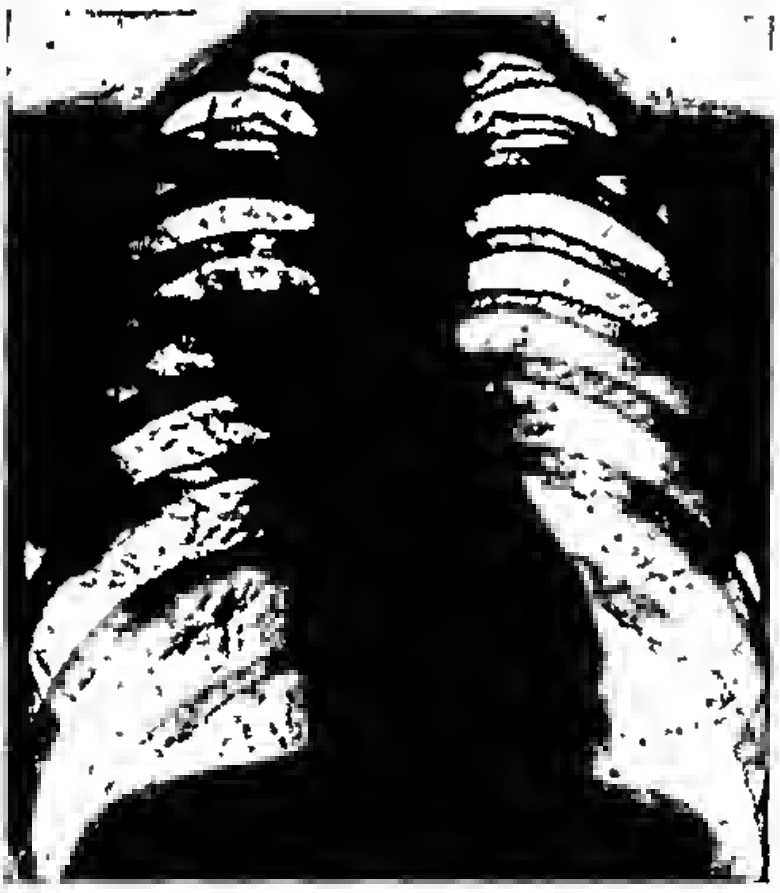

FIG. 53. Recovering partial pneumonia of right upper lobe.

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This condition is quite unlike the results of the "pneumonia of the new-born," because here atelectasis is a preceding entity; the lobules never opened at all, which is necessarily a different condition from acquired collapse. It is also quite unlike so-called "post-operative pneumonia," which is not pneumonia, but a purely bronchial disease. A sudden thick, sticky exudate from the wall blocks the lumen, with the same dramatic effect seen with an inhaled foreign body. Residual air cannot be renewed; the parenchyma collapses, and there is no obstacle to the action of the intra-pleural pressure on a lobe that perforce must shrink to its main bronchus at the hilum. Screening the patient or taking two films, one on complete inspiration and the other on complete expiration, will show us what never happens in ordinary pneumococcal pneumonia: a swing of the collapsed lobe, the heart, and the mediastinum, with each inspiration, to the affected side. There will be some recovery with each expiration, but still a displaced mediastinum, which will be shown by the positive sterno-mastoid sign even if it be impossible to demonstrate by the position of the apex beat. If the glutinous material is not cleared out by postural drainage or bronchoscopic suction the consequences can be very serious. Organisation can lead to every stage of bronchiectasis, the same end-result so often seen in the "pneumonia of the new-born." To this last condition the name "congenital bronchiectasis" may be applied; with its atelectasis, present from birth, there is often mal-development of bronchi, so that cysts are frequently found, and these the superimposed infection turns into sumps of septic material to add to the bronchiectatic end-result.

From what has been said of the pathogenesis of pneumonia the possibility of localised forms can be easily envisaged. The organisms travel some way along the main bronchus before piercing the wall of one branch. This is comparatively common in the pneumonia of childhood, which gives in one of its forms the so-called "hilar pneumonia," by involving the posterior horizontal branch of the right lower lobe bronchus. It is not uncommon to find in an adult that only the pectoral or the axillary branch of

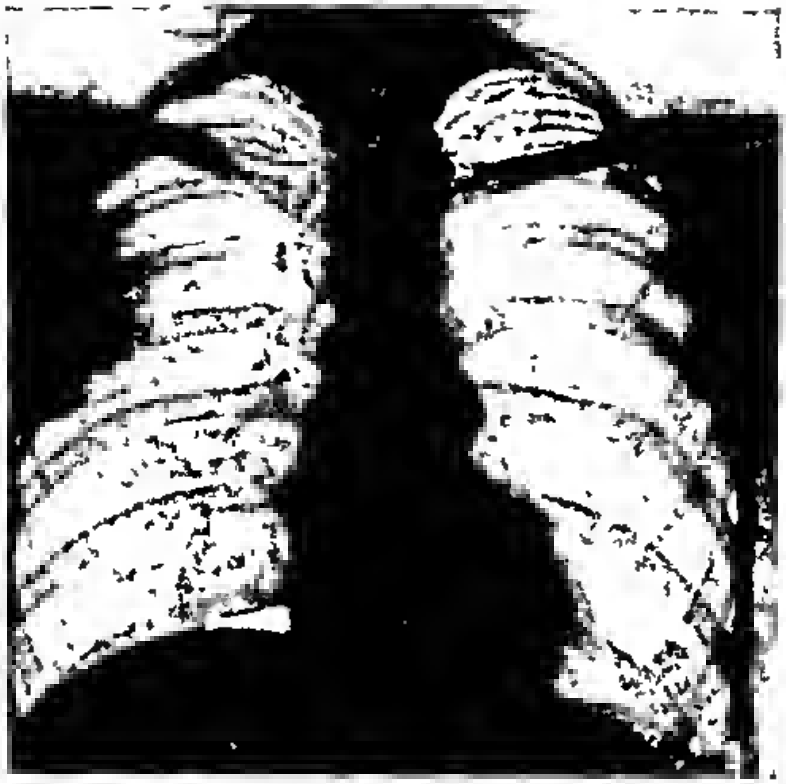

FIG. 52. Pneumonia of the axillary branch of the right upper lobe bronchus.

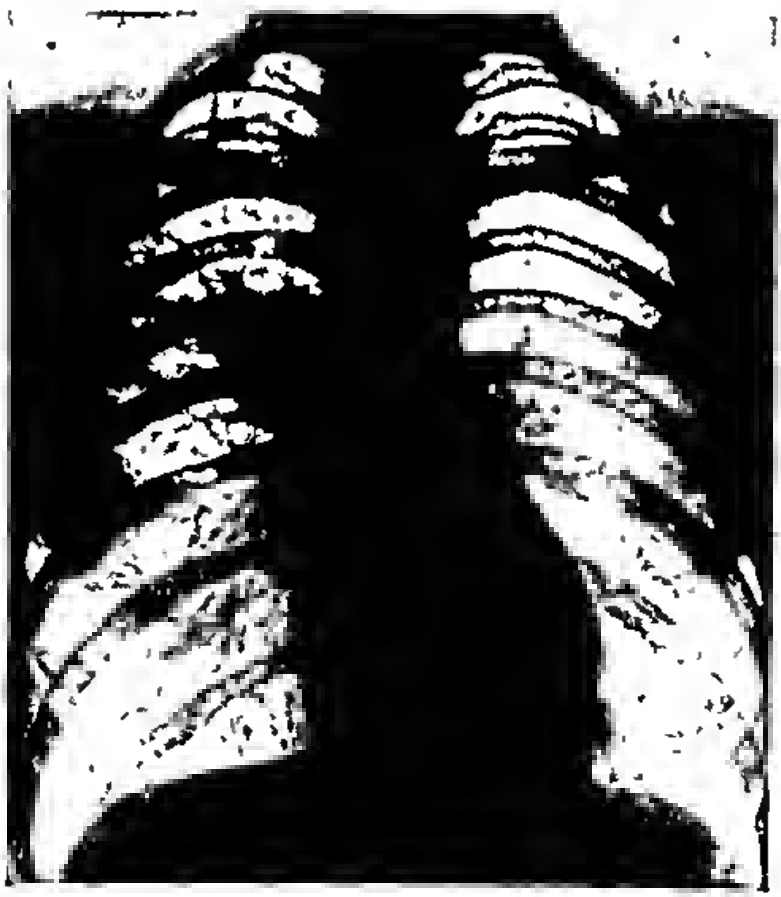

FIG. 53. Recovering partial pneumonia of right upper lobe.

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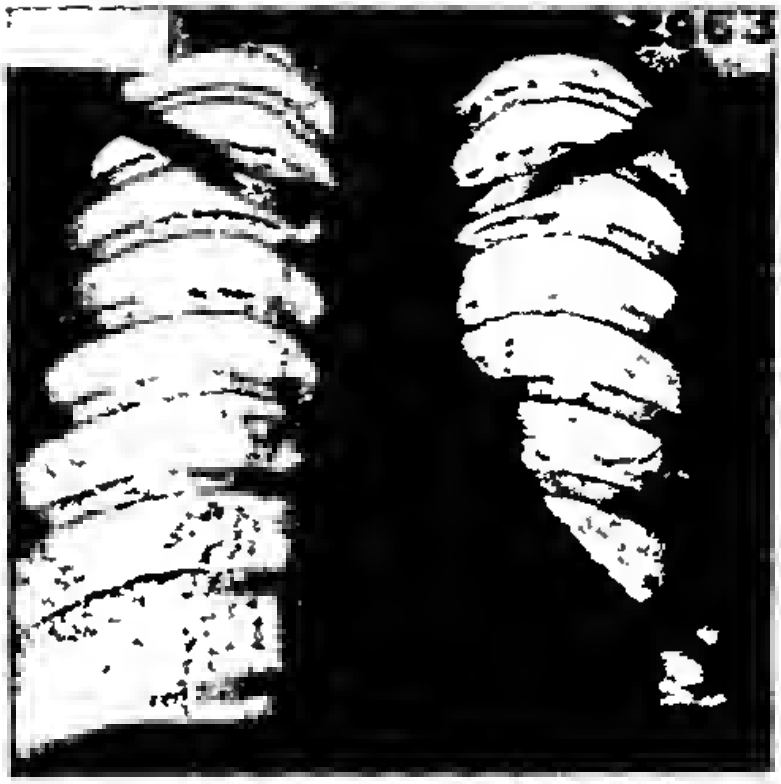

FIG. 54. Filling of left costophrenic angle in recovering pneumonia.

FIG. 55. Lateral view of fluid in main fissure producing "tenting"

the right upper lobe bronchus has been affected, so that we see on the film that triangular shadow of the cone of parenchyma already referred to in the section on applied anatomy (see Fig. 9, p. 6 and Fig. 52). The development of the disease is just the same as in lobar pneumonia: it spreads outwards from the point at which the bacilli penetrated the bronchial wall, so that all the segment of lung supplied by that bronchus is involved.

The shadow cast by these localised pneumonias is never really like that of tuberculosis with which it is usually considered in differential diagnosis. Both may give a triangular loss of translucency in the outer axillary region of the right upper lobe, the pneumonic one being due to involvement of the axillary branch of the main bronchus, while the tuberculous one is due to a tuberculous deposit in the periphery of the outer base of the upper lobe: that is, commonly just above the peripheral end of the lesser fissure. Both cause thickening of the fissure. Both can show abnormal films for a long time after onset, because localised pneumonia tends to heal by repair rather than by resolution. If, however, we look critically at serial films, we shall find that the tuberculous shadow always has its main continuing density against the periphery, as it is essentially a parenchymatous disease; the bronchi are involved only secondarily at all stages. This is not so with the localised pneumonia healing by repair: the main density is now at the apex of the triangle, that is, round the supplying bronchus at its entrance. Secondary involvement of bronchi in tubercle is at the site of the deposit; there the bronchi dilate steadily until, in most cases, definite cavitation is seen.

Another shadow that may take considerable time to clear after pneumonia is that of the exudate on the pleura, especially if the interlobar pleura has been affected. In all cases there is a true pneumonic exudation on the visceral layer, and this causes adhesion to the inflamed parietal layer; movement by inspiration therefore causes sharp, stabbing pain. Liquefaction usually leads to rapid absorption, but we have already noted how absorption may take a long time in the costo-phrenic angle and in the interlobar fissure, so that we get filling of the costo-phrenic angles and tenting of the diaphragm, that are permanent, but of no clinical import. In the same way the lesser fissure may be thickened for a considerable period after a partial pneumonia (see Figs. 54 and 55).

Broncho-pneumonia

Broncho-pneumonia, as already stated, may be considered a consisting of localised patches of pneumonia, confined by th strong resistance of the perihronchial connective tissue, whic limits the disease to bunches of lobules. It is commonly bilater.. from its onset, hut it may be completely unilateral, or develo on one side later than the other. It is usually in the lower lobes hut is not uncommon in the right middle lobe.

Its progress can be followed on the postero-anterior film, which demonstrates shadows that begin near the cardiac border and increase in size and in spread along the bronchial distribution, so that they go downwards and outwards in lower lobe involvement, and almost horizontally outwards in middle lobe disease (see Figs. 56 and 57). The involved areas show the same homogeneous losses of translucency as we saw in pneumonia, only now they are in their greater densities triangular in shape, particularly in the middle lobe, as the lobules are pictured along the side of their cone-shaped broncho-pulmonary segments (see Fig. 58). The lobules involved by pneumonic exudate are never entirely discrete, because they have round them those showing the first inflammatory, non-specific, catarrhal exudate. There is no shift of the mediastinum.

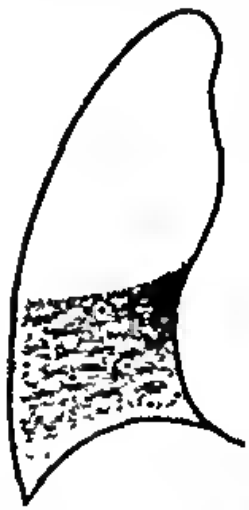

Fig. 57. Broncho-pneumonia.

The picture is therefore in two characteristics unlike that of broncho-pneumonic phthisis. Firstly, this disease is fibro-caseous and will show clear-cut areas of acinar collapse among others with complementary emphysema, in spite of the fact that both conditions may have surrounding œdematous shadows. This will be quite evident if we look at the films at some distance from the viewing box, when the shadows of streptococcal broncho-pneumonia will be no more evident, hut those of tubercnlous hroncho-pneumonia will become much more clearly cut. Secondly, there will always be some collapse, and therefore some displacement of the mediastinum, with phthisis, and if this is not clear to X-ray it will be to physical examination.

The physical findings can be explained hy the pathogenesis and the X-ray picture. We shall find patchy areas of silence

FIG 56 Bilateral lower lobe broncho pneumonia.

FIG 58. "Contact" film of right lower lobe in Fig 56

[To face p. 80.

over lobules filled to distension with pneumonic exudate, and immediately the disease is fully developed we shall get signs of this exudate entering the terminal bronchioles. The lobe involved is never, of course, completely silent: there are parts which have escaped altogether, and others where only inflammatory exudate occupies lobules still maintaining some airway. If we listen carefully we hear rather bubbly râles in the mid-third of inspiration, because this exudate is discharging into terminal bronchioles (see Fig. 51, p. 77). This is a distinctive stethoscopic finding throughout the clinical illness, the only difference being that the râles get more marked as the pneumonic exudate liquefies.

The dullness will naturally be much less than that of pneumonia as the involvement is patchy, but there will be distinct lack of movement laterally in cases where the lower lobes are diseased.

By reason of the much more intense connective tissue reaction many cases proceed to small areas of atelectasis. The increased tissue naturally gathers round the terminal bronchioles of the affected lobules. This interferes with the natural movements of the bronchioles in response to respiratory action; that is, their muscle fibre action is inhibited. Their elastic tissue reaction, which is dependent on the stretching of the alveoli to residual air, is already interfered with, since the alveoli are fixed at full expansion by the exudate in them. When the disease tries to resolve, these bronchioles are therefore caught at a distinct disadvantage. They have no time to recover from this initial irritation of their surrounding connective tissue before they are flooded by lymphocytes and plasma cells from the exudate, now trying to escape from the alveoli as resolution sets in. We have now irritation of the inner wall of the bronchioles added to their connective tissue irritation on their outer walls. They are, therefore, all too easily blocked, so that alveoli that have managed to empty go on to collapse, while those still containing exudate go on to organisation (see Fig. 46, p. 65).

We can see, therefore, the inherent danger of too early movement, perhaps advised by the belief that modern methods of treatment, by reducing temperature, have brought resolution. If this is dangerous in lobar pneumonia it is much more dangerous in broncho-pneumonia. If the bronchioles do not empty, their walls weaken, and the way is open to bronchiolectasis and later to bronchiectasis.

This is especially possible in right middle lobe disease. We have already noted that its main bronchus goes out almost horizontally from the right main bronchus. It is therefore difficult to drain. If it goes on to blockage in its terminal bronchioles we have a slowly changing picture and accompanying changes in physical signs. The areas that on previous distant viewing merged with their surrounding catarrhal, inflammatory exudate, now stand out with the clarity of broncho-pneumonic phthisis, with which we may confuse them because of the accompanying shift of the mediastinum. We shall note, however, that they are confined to lateral densities in one area of the lower mid zone, as against the widespread distribution of tuberculous broncho-pneumonia, and that they have always a much more exaggerated perihronchial fibrosis. A lateral film would show us how much more the lesser fissure shares in this fibrosis than it does in phthisis, and, of course, the general condition, and the lower and less-swinging temperature will be a further aid. Both conditions will of course give us the crepitations of fibrosis, but they will be much more localised, and definitely coarser and more insistent in complicated streptococcal broncho-pneumonia. Many cases seem to go on to simple fibrosis in the lower mid zone: indeed the fortunate patient may escape further clinical upset; but many go on to slowly increasing bronchiolectasis and to established and progressive bronchiectasis. This is a common end-result, long afterwards, by superimposed infections.

The patient may for a long time be unaware of what has happened. Mass-radiography has shown that many apparently completely recovered cases of middle lobe broncho-pneumonia have been left with much fibrosis. The X-rays shown in Figs. 59 and 60 are an example. The first demonstrates a loss of translucency in the right lower mid zone. The patient had signs of fibrosis in this area. The lordotic picture shows how the middle lobe has shrunk very considerably. Lipiodol later proved the presence of bronchiectasis.

An important point to remember is that every case of broncho-pneumonia causes marked increase in hilar shadows because of the intense hyperæmia of its resistance; the hilar glands also swell with œdema. This hilar increase takes a long time to clear up, and this is especially noted with the usual type of scoliosis which brings the left hilum into prominence. There is the possibility of a grave diagnosis of localised glandular disease, but the

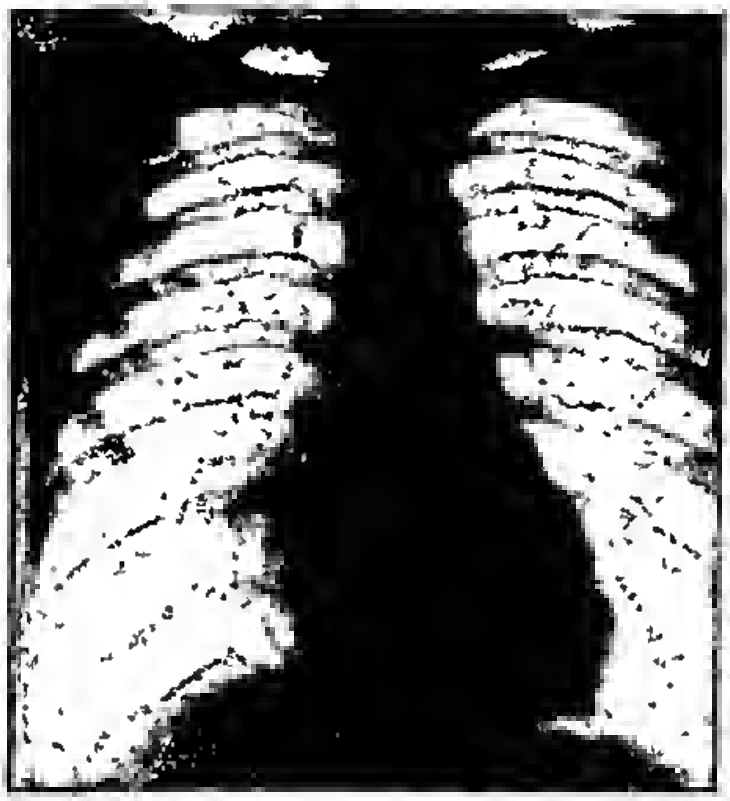

FIG. 59. Fibrosis following right middle lobe broncho pneumonia

FIG. 60. Lordotic view of same patient as Fig. 59.

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swelling is seldom of the extent of Hodgkin's disease, and the swing of the Pel-Ebstein fever, and the blood count, will be further aids in doubtful cases. The enlargement remains long after the patient is clinically well.

CHAPTER XIV

ATELECTASIS

In dealing with this condition, which may be defined as collapse of parenchyma through loss of residual air, we shall confine our discussion to the acquired form due to obstruction of the lumen supplying a broncho-pulmonary area. This may occur by something inside the bronchus, such as a growth or inhaled foreign body, blood, mucus or granulation tissue; or by something pressing on the wall of the bronchus, such as an enlarged gland or tumour formation. As we saw in our studies of applied anatomy, and in the description of post-broncho-pneumonic atelectasis in the right middle lobe, the size of the affected cone depends on the size of the bronchial lumen. The same we shall find to be true in the secondary results of blockage of the terminal respiratory bronchiole in adult phthisis, where granulation tissue enters it from the primary acinar deposit. If a main bronchus is occluded, as in "post-operative pneumonia," then the whole lobe collapses, since the residual air to every terminal alveolus is absorbed.

In such a condition oxygen is first absorbed by the capillaries in the alveolar walls, and we get on the film a fine loss of translucency. We cannot see this taking place in the small atelectatic areas of bronchiectasis or complicated broncho-pneumonia, but we can follow it quite easily where a whole lobe is shutting down, and we shall always find this slight homogeneous shadow beginning in the least supported areas of parenchyma. Thus in the upper lobe it will occur first in the outer apex, and in the lower lobe along the anterior basal margin; we saw the reason in discussing structural support under our considerations of applied anatomy; these areas are dependent almost completely for their normal position on their residual air, and have but little support from their scaffolding of connective tissue. Later the nitrogen is absorbed, and the shadow, while still homogeneous, becomes definitely more dense.

Up to the time when such density covers the whole of the affected lobe it may be possible, in those cases that are due to carcinoma of the bronchus, to differentiate on the film the heavy opacity of the actual growth near the hilum, next to it the slightly

less opacity of those areas already completely collapsed, and towards the periphery the finer loss of translucency of early collapse. We have here a characteristic "whitewash brush" film (see Figs. 61 and 62). It looks as if the brush had been applied heavily over the point of entry of the bronchus, and then, as it was swept outwards towards the periphery, had been pressed with continuing less force. The apex of the broncho-pulmonary area is densest; its base least dense. Later, as the lobe becomes smaller by shrinkage, and is followed up by its proximal lobe which develops, *pari passu*, its complementary emphysema, the interlobar fissure rises, and the density becomes throughout equal to that of the causative growth, which indeed in most cases has by this time extended by its cells, or its mucinous exudate, into a considerable portion of the surrounding parenchyma. In many there appears by now the further complication of abscess, bringing a more or less central area of translucency.

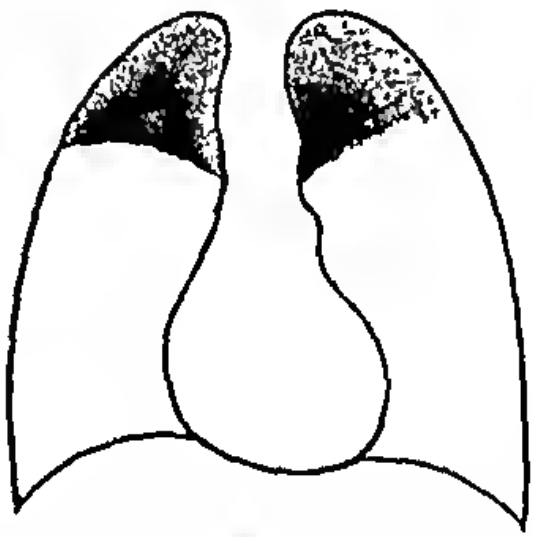

Fig. 61. Carcinoma left upper lobe bronchus, compared with tuberculous pneumonia of right upper lobe.

From the beginning there will be lack of movement, and dullness will deepen with increasing involvement of the growth. The mediastinum will drift to the same side. We shall find the evidence of shrinkage in alteration of the normal lesser fissure or the appearance of the greater fissure on the postero-anterior film, and in the change in blood supply striations in those parts of the lung that are blown out by complementary emphysema. Lateral films are of particular assistance, as we have already noted, in all conditions effecting change in size and shape of lobes.

It follows that the dullness of collapse must be accompanied by the tympany of emphysema. This is why broncho-pneumonia and its complication of terminal bronchiole blockage never give over-all dullness, and why with the dead dullness of the collapsed right upper lobe we find marked tympany in the compensatory distension of the middle and lower lobes.

Bronchial breathing may be very marked just short of the

actual growth at the origin of the affected bronchus. Added sounds inside the collapsed lobe do not present invariably in the upper lobes unless there is damming back of secretion and the complication of abscess is superimposed. Once the extension of growth begins to press on the bronchus to a lower lobe we get, beyond this second bronchus, a very high-pitched musical sibilus from air passing the obstruction, as in the production of an instrumental whistle. Its appearance is of grave import.

The most constant sign of collapse in all its forms is the sternomastoid sign, which is always positive on the affected side. In the difficulties of differential diagnosis it is a much more reliable finding that any other single aid, even including comparison of films on complete inspiration and complete expiration. We have already noted how the trachea reacts to changes in normal lung elasticity, the shrinkage of the affected part leaving the remaining normal tissue open to both increased pull on the same side and increased push from the other. While this tracheal reaction is constant, the position of the heart is variable, for as we saw, its greatest weight is in the lower mediastinum, and it will react only to lower zone disease or complete unilateral disease. Even then it is unwilling to respond to anything minor in force; it will swing on its long axis and can be pushed backwards by the emphysema of the opposite side before it has moved bodily and laterally to the affected side. While it must certainly always be located by palpation and by stethoscope, the position of the apex beat in collapse may be a misleading sign. Even to screen examination the heart may show no definite shift with inspiration if the diseased area is above the origin of the aorta. These tests we shall see applied in more detail in considering atelectasis of individual lobes.

Upper Lobe Atelectasis (see Fig. 61)

The commonest cause is carcinoma of the bronchus. It is here we see best demonstrated the "whitewash brush" effect on the film, although the very first evidence of interference with residual air may appear in a fine loss of translucency in the outer infraclavicular region. Quite early we can see the accessory signs in shift of the trachea and the mediastinum to the affected side, and in the upward shrinkage of the interlobar fissure on to which the thin triangular lingula retreats steadily. The interlobar fissure becomes more and more evident because the rays are passing

through more and more tissue; it has added to it all the tissue of the retracting lingula of the lobe. These findings are always more easily recognised on the right as against the left side, as the shadow of the upper right cardiac border is made in great part by the movable superior vena cava, usually less well confined than other mediastinal structures by the surrounding areolar tissue. The heart does not move much in the lower two-thirds of its extent, and so there may be no appreciable change in the position of the apex beat to palpation or auscultation even with considerable shrinkage of the upper lobe.

It is quite possible that in its early stages the film can resemble that of a developing pneumococcal pneumonia, although temperature, symptoms, and the examination of serial films should help us to avoid misinterpretation, and we know that an uncomplicated pneumonia does not produce mediastinal shift. A lateral film will aid us because we shall see the retraction of the lobe on to the inner end of the lesser fissure in a manner never displayed with developing pneumonia. Further, we shall find that while pneumonia gives a dullness both anteriorly and posteriorly, that of collapse is always anterior and below the inner end of the collar-bone, *i.e.*, at the site of origin of the bronchus.

The difference in site of dullness is of distinct help if the diagnosis is against upper lobe "pneumonic" phthisis; here the dullness is posterior, as tubercle in adult form is a posterior disease, inclined to attack parenchyma supplied by dorsally-inclined branches of the bronchial tree. Again, tuberculosis is a parenchymatous disease in its pathogenesis, not a bronchial disease, and the greatest density in its picture is noted below the middle third of the collar-bone and not over the site of the entrance of the main bronchus.

Right Middle Lobe Atelectasis

We noted when dealing with right middle lobe broncho-pneumonia that the dangers of inefficient bronchial blockage are considerable, owing to the angle at which the main lobar bronchus comes off the right bifurcation. Post-pneumonic collapse by viscid sputum is very common, and occurs in many cases with perhaps little immediate, but grave after-effects. It can come on quite suddenly or very slowly. In the latter case the pain has a characteristic position along a band below the right nipple, coming in waves of intensity up to a pitch of severe discomfort.

The postero-anterior film gives a shadow outlining the lobe, heaviest along the line of the lesser fissure and lessening to an area of translucency above the diaphragm in spite of the fact that this muscle rises towards its position of expiration against the emphysema of the lower lobe (see Figs. 63 and 64). The lateral film is characteristic and unlike that of any other disease. It is almost always of the "whitewash-brush" type, dense at the entrance of the bronchus and fading toward its base. The npper side of the triangle is along a line beginning at the junction of the middle and lower thirds of the mediastinum and going horizontally outwards to the posterior aspect of the sternum. The inner side of the triangle goes downwards and ontwards to a point near the middle of the diaphragm (see Fig. 65). The case demonstrated in Fig. 64, was one of collapse due to benign tumour of the middle lobe bronchus: the growth was removed through a bronchoscope.

The lateral film of collapsed middle lobe can cause difficulty in diagnosis as against effusion in the lesser fissure. The upper border of the fluid is seldom straight as in collapse: it is inclined to bulge upwards in convex fashion, and even if it is convex downwards, because the fluid is now encysted, we do not get evidence of emphysema in other parts of the lung. Physical signs should also help. The fluid will be encysted, dense, and restricted in area before it causes drift of the mediastinum towards it, and it will not give us the marked tympany of complementary emphysema which is bound to accompany collapse (see Fig. 66).

Fig. 67. Pleurisy secondary to abdominal disease.

The swing of the heart to the affected side by inspiration is more marked in middle lobe than that found in upper lobe collapse. The two characteristics to physical examination are the positive sterno-mastoid sign, and dullness which is anterior in the lower mid zone. The emphysematous lower lobe gives definite posterior tympany.

The only other condition giving such marked anterior dullness is fluid in the main interlobar fissure, but its films antero-posterior, lordotic and lateral are entirely different, the first showing a loss of translucency sitting above the diaphragm like that

FIG. 62. Carcinoma right upper lobe.

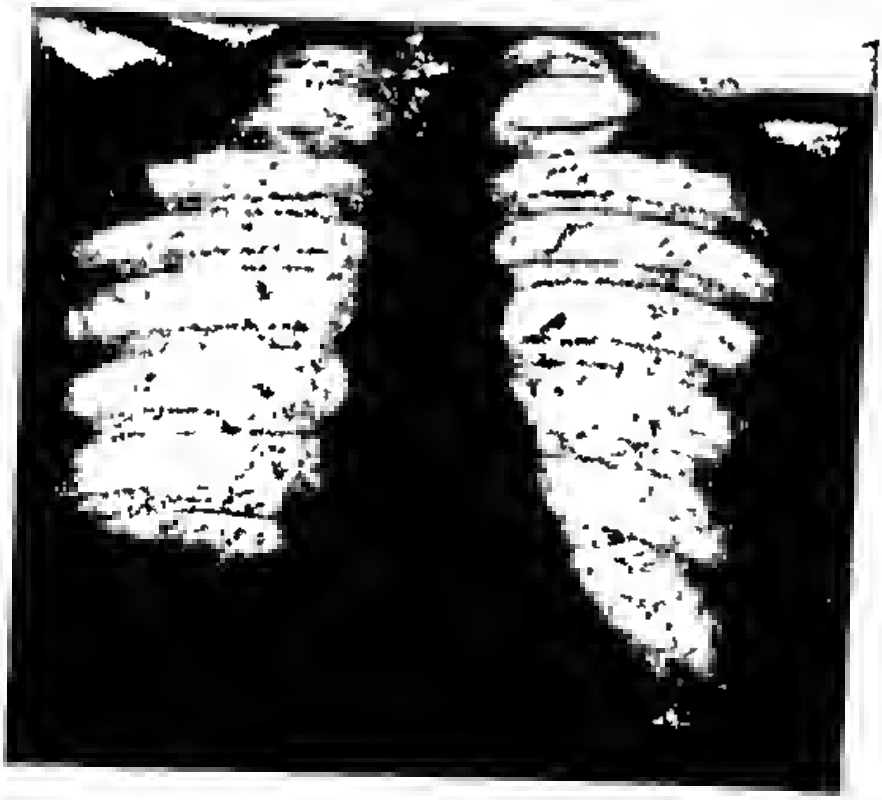

FIG. 63. Collapsed right mid-lobes from broncho-pneumonia.

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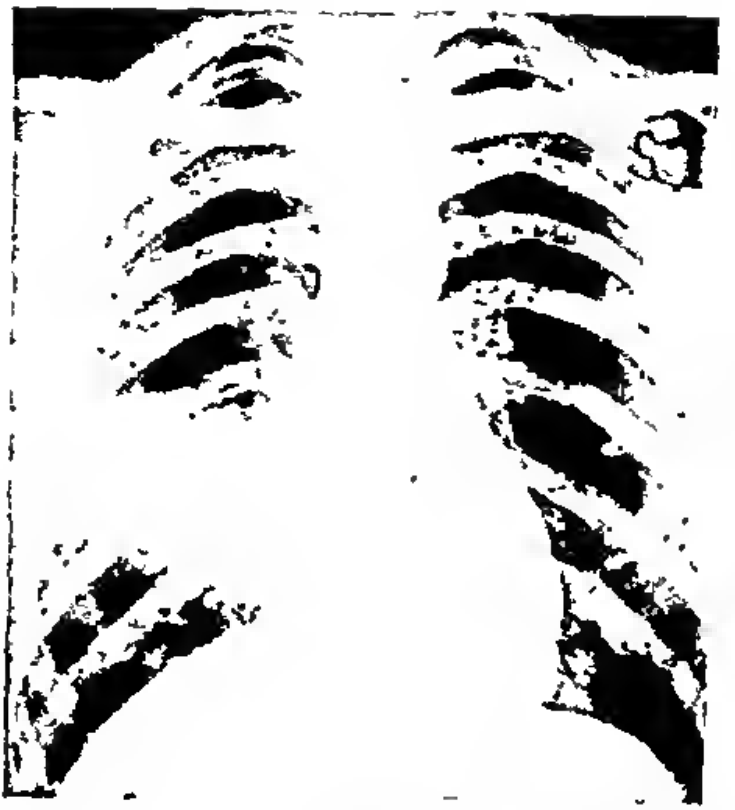

FIG. 64 Collapse right middle lobe, by innocent intra-bronchial growth.

FIG. 65 Lateral view same case as in Fig. 63.

FIG 66 Lateral view an interlobar effusion.

FIG. 68. Fluid in left interlobar fissure.

[To face p. 80.

of pleurisy secondary to a sub-phrenic abscess, the lordotic a tent-like shadow with its base over the diaphragm, and the lateral a density between the cardiac shadow and the diaphragm (see Figs. 67 and 68, and Fig. 55, p. 79).

As already noted, many cases of post-broncho-pneumonic collapse of the middle lobe can pass unrecognised at the time of the clinical illness, as there is much less upset in pulse and temperature than occurs in the average case of lower lobe "post-pneumonic" collapse. Only considerably later, after what then appears to have been a rather protracted convalescence, does the patient complain of dragging pain; full investigation, including lipiodol injection, commonly shows considerable dilatation of the bronchi. Bronchiectasis is by no means rare, sometimes by super-imposed infection, but often only by the continuing effects of the collapse, which has led to abscess formation by the damming back of the exudate.

This type of case often presents one of the most difficult problems of differential diagnosis, because the abscess almost always produces the complication of a frank pleurisy, with the danger of consequent empyema. We are now presented with a condition which is all too easily read as a pleural effusion. Fortunately the tracheal displacement in response to atelectasis is always more marked than the pressure of the fluid; in other words, the sterno-mastoid sign remains positive on the right. The writer has seen several cases where this was the main indication to the true diagnosis. It is of course taken for granted that all other clinical aids, including needling, will be employed for every ultimate diagnosis.

Lower Lobe Atelectasis (see Figs. 69, 70, 71 and 72)

The commonest types result from pneumonia, broncho-pneumonia, "post-operative pneumonia," and new growth of the bronchus. Of these the most sudden in onset, and the most massive in result, is the "post-operative," due, as we have already noted, to the formation of a glutinous exudate on the wall of the main bronchus. The pneumonic and broncho-pneumonic ones are usually slower in development, and therefore approximate to the slower shrinkage seen in bronchial carcinoma.

The characteristic shadows of the preceding pneumonia has been described, and the changes in broncho-pneumonia which goes on to lobular and lobar collapse have been detailed. In

pneumonia there has usually been considerable progress town recovery, with clearance of the homogeneons density, from crisis in temperature, before the complication occurs. Wl collapse sets in by blockage of the main bronchus by a plug sputum, the film goes throngh a series of nlterations similar those seen in hoth post-operative pneumonia nnd bronch carcinoma : that is, density begins again at the hilum.

It is essential to remember that the same picture can resu from abscess and from tuberculosis, at the npex of the lo (see Fig. 15, p. 8). Radiography can fail altogether to giv the true cause of what is seen. It may have been possible t follow the developing collapse from a rounded opacity close t

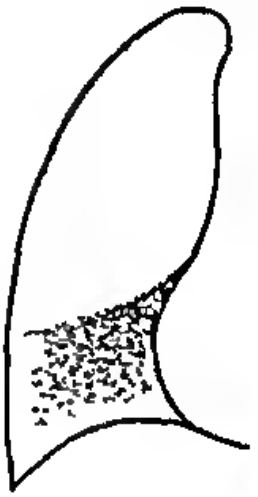

Fig. 69. Lower lobe atelectasis, Stage I.

Fig. 70. Lower lobe atelectasis, Stage II.

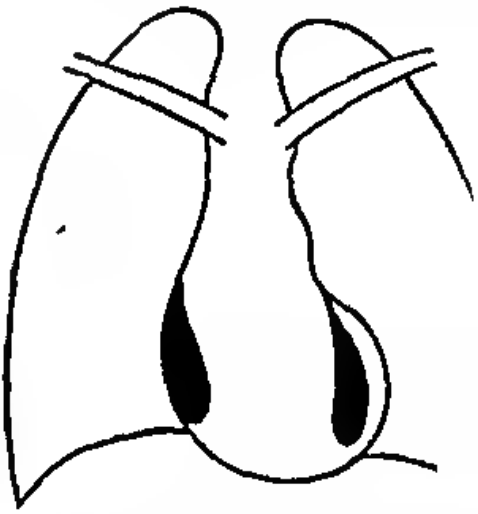

Fig. 71. Lower lobe atelectasi Stage III.

the hilum, hut usually one sees only a partial collapse in the fir X-ray, the apex of the lung being already hidden in the hil... shadow. Full clinical and bacteriological investigation, and serial X-rays, must be considered in every case.

Three stages of lower lobe collapse may be detailed : early, half, and complete collapse. All three have the homogeneous type of shadow ; that is, it may alter in density but not in smoothness throughont its extent, no part of it, but only the whole, standing out on distant viewing, as the concurrent emphysema is outside the boundary of the affected lobe. All three stages have the same npper point to their loss of translucency. This lies at the upper limit of the ventricular or auricular shadow on the eardiae border ; left- and right-sided cases are similar in this apical hilar limit to their triangular shape.

FIG. 72. Right lower lobe collapse in second stage.

FIG. 73. Squat heart. Compare Fig. 72.

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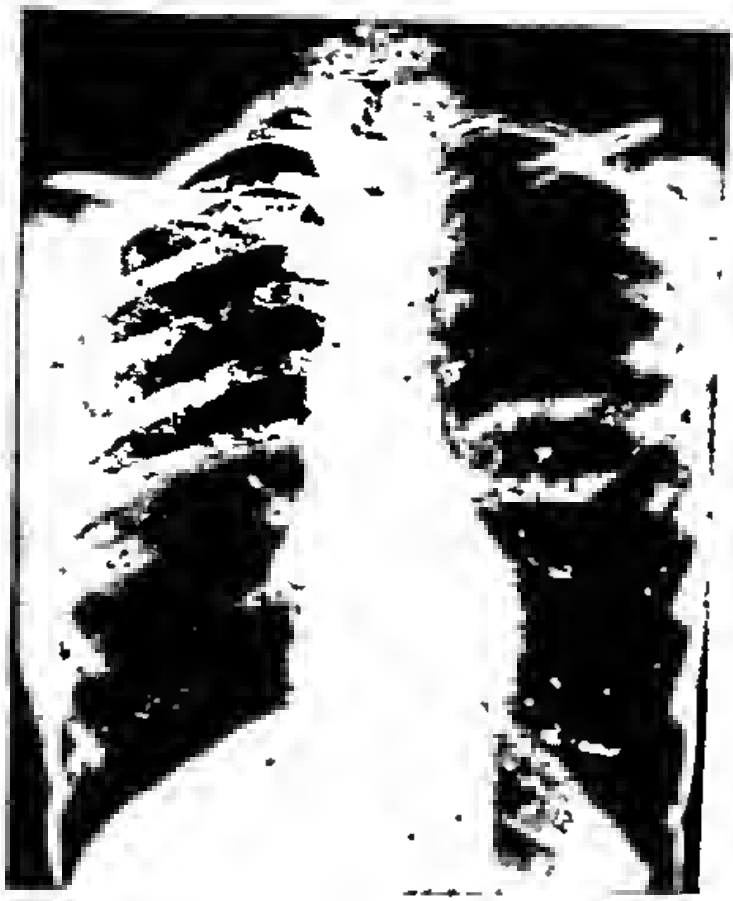

FIG. 74 Third stage collapse of both lower lobes.

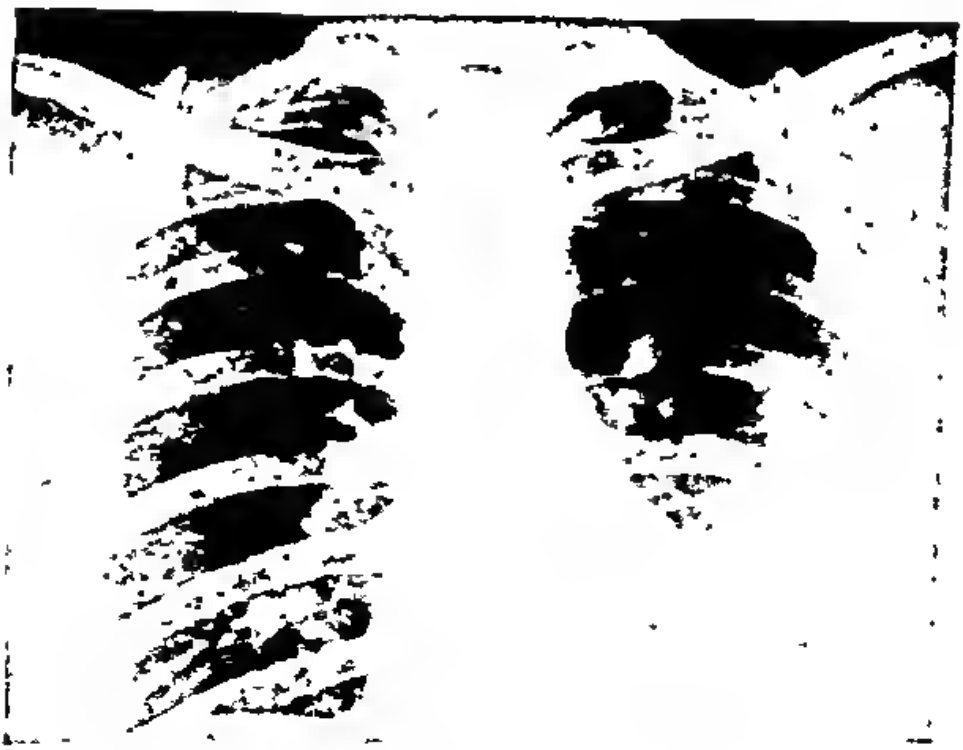

FIG. 75. Left lower lobe collapse with fluid—sternomastoid sign positive on left side.

The first stage shows a loss of translucency diminishing from this point downwards and outwards to the periphery, the upper border being made by the interlobar fissure. It will be realised from the anatomy of the lobe that this means that its apex has already undergone a considerable fall through shrinkage of the triangular lingula, and this we would expect, as the lingula, being thin, has less structural support, as well, necessarily, as less residual air for absorption. The blood vessels of the partially collapsed lobe are crowded towards the mediastinum, which is displaced to the side of the lesion. With this the most marked change in physical signs is the sharp tension of the sterno-mastoid on the same side; it was unaffected by uncomplicated pneumonia. At the same time we note that the dullness, which had been clearing both anteriorly and posteriorly, is now again marked posteriorly; this is because the lung shrinks downwards and backwards as well as inwards. The last two movements tend to make the heart swing on its axis: thus in right lower lobe collapse the right lower border recedes and the left lower border comes forwards and to the right. The apex beat may therefore be but little disturbed from the normal, and is not a reliable aid to the diagnosis. The swing of the heart on inspiration to the affected side will be quite evident to screen examination.

In the second stage the shadow is much more dense; its outer border goes downwards and outwards towards the mid-point of the diaphragm on the affected side. So dense is the shadow that it appears to be continuous with, and equal to, the heaviest density of the heart shadow towards its mid-point. There is therefore total obliteration of the cardio-phrenic angle. It follows that this shadow ought not to be confused with that of a "squat heart" in cases of left lower lobe collapse. True, the diaphragm is higher than normal in both conditions, due to lobar shrinkage in collapse, and to its being pushed up by abdominal fat in the case of the "squat heart," but the shadow is essentially different (see Fig. 73).

Moreover, there are further film and physical findings which are conclusive. Collapse, as we noted, must bring emphysema; in this case it is in the costo-phrenic angle, where we can see marked loss of normal lung markings on the film, while in the upper and mid zones we find the vessel-markings splayed out much more radially than in the normal. There will therefore be marked tympany in the axilla, off-setting the increasing

dullness in the lower inner third posteriorly, and we shall hear the blowing breath sounds from the bronchus over its point of entry near the eighth or ninth dorsal vertebra, because the collapsed lung is acting as a sounding board.

The third stage is shown on the film as a very dense area, heavier than that of the cardiac border, rather like a collapsing toy balloon with its neck attached at the upper cardiac border. On the right side its outer edge is usually concurrent with the right cardiac border on the postero-anterior film, while on the left side it lies within it (see Fig. 74). It is in such cases in particular that the complementary emphysema may be the only indication of abnormality. By this stage there is emphysema on the opposite as well as on the affected side, and this has an important bearing on physical findings. We have learnt earlier that the mediastinum is one continuous whole, not a structure with right and left sides. As one side of the thorax shrinks, therefore, the mediastinum drifts as a whole. In collapse of the right lower lobe with drift of the mediastinum to the right, there is also drift to the right of the left lower lobe, which by emphysema attempts to fill the vacated space. This it does by occupying the lower anterior mediastinum, and in doing so it must perforce overlap the anterior surface of the heart, pushing it backwards. We have therefore seen two reasons why the heart swings and goes backwards rather than move to the side of the atelectasis during conditions of respiratory rest. Herein lies the reason why we get, with complete collapse, anterior dullness in the affected cardio-phrenic angle; we are percussing the heart pressed back to be continuous in note with the much shrunk lower lobe. All over the remainder of the lung, and particularly in the lower axillary zone, we shall find marked tympany.

If, however, we screen the patient or take two pictures, one on complete inspiration and the second on complete expiration, we shall see the effect of the strengthened intra-pleural pull on the affected side, for the heart comes well over with each inspiration. This finding, combined with a tense sterno-mastoid muscle, will aid us in diagnosis, but we shall find the latter the better help if the patient has the complication of fluid in the pleural cavity, for this will undo the tendency to swing; it has brought the sub-atmospheric pressure nearer to atmospheric or above it. The muscle tension still remains; the pull on the trachea by the uncollapsed parenchyma is still present. If therefore we find a

patient who is dull all over the lower half of his chest, both front and back and in the axilla, on the left side, and still has a tense left sterno-mastoid, he cannot have uncomplicated pneumonia because pneumonia does not move the mediastinum; equally he cannot have left lower lobe pneumonia with pleural effusion because this would give a right sterno-mastoid sign, and so he must have a collapsed left lower lobe with pleural effusion (see Fig. 75).

CHAPTER XV

LUNG ABSCESS (See Fig. 76)

It is well at this stage to consider the pathology, X-ray appearances and correlated physical findings of abscess of the lung, as amongst its commonest causes are preceding broncho-pneumonia, more rarely pneumonia, and now and again, in one of its forms, atelectasis from main bronchial blockage.

To get a clear picture of the most usual process, it is best to envisage it as due to aspiration of septic material along a bronchial branch into the lobules of the broncho-pulmonary segment supplied by that branch. This is not the same thing as blockage of the branch, which is really a complication, or an entirely different entity. Unless we grasp this fundamental point in pathogenesis we shall be unable to understand why one abscess clears completely and leaves no ultimate damage to the parenchyma, while another goes on to a chronic cavity and to resulting bronchiectasis; and why the physical signs in the two processes are quite different. The essential difference in microscopical findings is that in the first elastic tissue is not destroyed; it is destroyed completely in the second, bringing atelectasis and organisation of lobules, and thence infecting the blocked bronchial wall to induce all the changes up to bronchiectasis. This is the reverse process to what we saw in advancing primary bronchiectasis, which produces abscess by extension of sepsis through the penetrated cartilage of the bronchial wall. In other words, as we shall learn later, this complication of aspiration is not unlike the pathogenesis of adult tuberculosis in so far as it is a progression from lobule to bronchus.

As the condition is primarily due to aspiration it follows that gravity will favour its onset. This is why it is common in lower lobes as against upper lobes, is more liable to occur on the right than on the left side, and why also it is peripheral rather than central in position. It can, however, be caught easily in its outward journey in any area supplied by a backward-going bronchial branch. This is one reason why we paid especial attention in our study of applied anatomy to the dorsal branches of the right bronchial tree (see Fig. 12, p. 7). Thus a very

common site for abscess in broncho-pneumonia is the segment supplied by the posterior horizontal branch of the right lower lobe bronchus as it hooks over the upper and posterior part of the lobe. The abscess shows on the postero-anterior film close to the cardiac border. It is the resulting "hilar" shadow of many cases of right lower lobe pneumonia, and presents a circular or oval opacity on the postero-anterior film, being photographed through the midline of the cone of infected parenchyma. A lateral film will throw the shadow into its true position in the apex of the lobe. We have already noted, in our consideration of lower lobe atelectasis, that an abscess in this position usually causes partial collapse of the lobe, and that the X-ray may be no different from that of tuberculosis in the same bronchial branch.

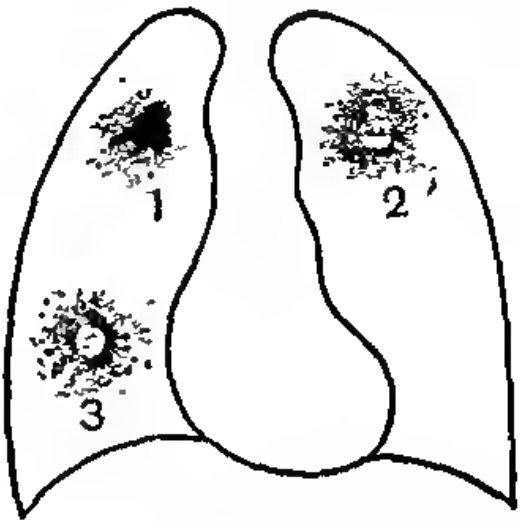

FIG. 76. Lung abscess. Stages in development described in text.

Such a complication of pneumonia or broncho-pneumonia is, of course, quite different in pathogenesis from the small multiple abscesses that can occur in the connective tissue in bronchiectasis; in this case the septic material has entered a blood vessel opened up in the destruction of the elements of the wall as sepsis proceeds to the parenchyma.

The peripheral aspiration brings the majority of abscesses close to the lung surface, so that the pleura is usually affected from the onset. This is why the earliest diagnostic sign is so often a coarse pleural rub, at first much more extensive than the area of underlying dullness. Fortunately the irritation causes local thickening and adhesions, especially in pneumococcal cases, so that empyema by rupture and extension into the pleural cavity is comparatively rare. Such adhesions may, however, form between adjacent lobes, allowing the overflow to pass into the fissure and cause interlobar empyema. Pain is not common with the pleural involvement, as there is not the large area of parietal pleura inflamed and pulled on, which we find in lobar pneumonia.

At an early stage the abscess is not easily defined post-mortem for the same reason as it is not of equal density in its rounded area of loss of translucency on the film. It is heaviest in its centre, as here are the lobules nearest the bronchial branch along which came the aspirated material. Outside these are the lobules which have been irritated in their alveolar walls so that they contain catarrhal, inflammatory exudate, giving the fine loss of translucency of œdema. The homogeneity of shadow is a diagnostic X-ray feature, and we shall see it remains until cavity formation occurs in those cases that go on to blockage of the bronchus. We see, therefore, that it is of the same consistency as the shadow of lobar pneumonia and of the individual shadows of broncho-pneumonia, all three having in common material which is filling lobules and replacing their residual air. Putting this in another form, the previous lobar shadow of pneumonia is now in a more or less circular, localised area ; the shadows of broncho-pneumonia are now confluent in a larger single area.

Sometimes abscess involves a considerable area, showing no special denser, central region, but only a wide-spread area of homogeneous loss of translucency. There is no particular definition, such as appears in lobar pneumonia, making out a definite lung segment (see Fig. 77).

At first the area must be silent, apart from its complicating pleural rub. It will cause no shift of the mediastinum, as its material has the same physiological effect on lobular elastic tissue as the residual air it replaces. There will be no sterno-mastoid sign, nor will it be found in the usual following stage of the abscess which evacuates voluntarily. This stage is often referred to as a cavitation because the picture on the film is misread ; there is no real difference except in extent between this second stage and the second, evacuating stage of a broncho-pneumonic single lobular affection. Naturally the lobules nearest the draining bronchus evacuate first, so they make a central translucency within a circular homogeneous shadow made up of those lobules still undrained. This ring shadow has no components of organisation or collapse ; it does not alter within itself or stand out against any surrounding emphysema with distant viewing as a tuberculous cavity wall does (see Figs. 78, and 90, p. 111). Stethoscopic findings bear this out ; we hear just the same râles as we heard in broncho-pneumonia in the mid-phase of inspiration, indicating lung material entering the bronchus, but naturally they are in-

FIG. 77. Lung abscess.

FIG. 78 Lung abscess.

[*To face p.* 96.

creased in number and coarseness as a bigger circumscribed area is affected.

It is necessary to repeat again, even if it appears redundant, that the usual lung abscess is not a cavity, as many students seem to visualise the process. It is merely, to all intents and purposes, a very localised pneumonia, filling lobules with exudate as pneumonia does, and differing only by the method whereby it was caused in these lobules. It distends them as pneumonic exudate did, and does not destroy their elastic tissue unless it cannot get out into the bronchus. Most patients evacuate the material voluntarily, at a stage when it contains no elastic fibres. Moreover, this method of cure never has with it any clubbing of the fingers; the septic material has not been dammed back.

Should, however, this evacuating material block the bronchus, the way is open to serious consequences, from cavitation to bronchiectasis. The first hint can often be got by the film examination; the exudate, now fluid and thin by the proteolytic enzyme of its polymorphs, is dammed back, and we see a level within the diseased area, confined by the circular or oval shape of outlying lobules which are still filled with large mononuclears, phagocytes and polymorphs. Should this not be evacuated by drainage, by conservative or surgical methods, then repair, and not resolution, will take place in these surrounding lobules. They cannot find an exit for their material content, and slowly fibroblasts will enter them, organisation and collapse will follow, drag on the bronchus be the inevitable result, and bronchiectatic cavitation by advancing sepsis from lobules to bronchus is the final picture. The septic material now destroys the parenchyma, and the foul sputum contains elastic fibres. There will be an immediate reaction in the mediastinum. To all intents and purposes we have one single cavity of bronchiectasis. There we saw how sepsis in the bronchus extended through all the coats of the bronchial wall until it reached the parenchyma, and produced a cavity in the lung connected with a saccular "cavitation" of the bronchus. This is just the reverse process leading to the same end-result. The disease can now extend like any individual cavity in primary bronchiectasis.

The physical signs are now those of organisation, fibrosis and atelectatic collapse. By destruction of elastic tissue we have drift of the mediastinum with a positive sterno-mastoid finding; to the stethoscope we have the crepitations of fibrosis; and the

coarse, metallic râles of bronchial dilatation and its connection with lung tissue are heard throughout the first and mid phases of inspiration. These are exactly the same added sounds as we found in advancing bronchiectasis.

Clubbing of the fingers of the septic type comes on rapidly with the blockage of the bronchus. If evacuation by efficient drainage is carried out the clubbing disappears quite soon; if it does not do so, then the observer should be doubtful if the lung condition is really cured. Many cases are shown by tomography to be going steadily, if slowly, forward to the stage of bronchiectasis in spite of apparent cure by operation. This is why there are so many so-called recurrences of lung abscess; they are not recurrences, but continuances of the same initial uncured lesion. We are now dealing with secondary bronchiectasis, not a lung abscess.

CHAPTER XVI

TUBERCULOSIS UP TO EARLY ADOLESCENCE

WHATEVER be its route of entry to the body, by inhalation or ingestion, the tubercle bacillus can always reach the lungs. When it comes in contact with any mucous surface it is ingested by a phagocyte, carried by it through the mucous membrane to enter the lymph spaces, and thence is conveyed to the lymphatic node or tissue draining this particular area. Its next stage on the journey is the venous blood and so the right heart is reached, and, through the pulmonary circulation, the lung tissue. Should it be held up in a small capillary the phagocyte pierces the wall to enter the surrounding lymphoid tissue, and there to form a focus of disease. In other words, whatever be the manifestation of the disease in the lungs from childhood to old age, tuberculosis is primarily a lymphatic disease. Its forms depend on the reaction of lymphoid tissue: it always attempts to drain along the lymph channels, which we saw in our consideration of applied anatomy are part of the protective system, together with the lymph spaces and the glands. It succeeds in most cases in the infections of childhood type, but its very evidence of acquired immunity, in its efforts to confine the infection to localised areas, can be the undoing of the infected subject as age advances, since the lymphoid tissue, so hedged about, becomes the breeding ground for the bacilli, and so itself breaks down. It is worthy of note that increasing age brings much increase in lymphoid tissue throughout the lungs: this is doubtless in great part an explanation of the widespread ravages of senile phthisis, which yet maintains such a resistance as to simulate only chronic bronchitis.

Although there are no sharp lines of demarcation dividing the disease into forms for different ages, it is convenient to describe its further developments under the headings of "Infancy and Early Childhood," "Childhood to Early Adolescence," and "Early Adolescence and Adult Life," as they show fairly defined types of response of the now infected organism to fresh infections.

Tuberculosis in Infancy and Early Childhood

The forms of the disease shown in this age group are miliary,

broncho-pneumonic and pneumonic. Closely interconnected in it, as in all age groups, are the three factors of :—

(1) The size of the initial dose and its repetition.

(2) The resistance of the body to these infections.

(3) The mode of spread.

By the method of initial lung deposit, lymphatic spread is commonest, small nodules tending to form in the peri-vascular and peribronchial lymph vessels. There is no doubt that this form can occur at ages long after childhood, in adolescent and adult life. The bacilli have, however, only to break into a blood vessel round which they are congregated to give miliary tuberculosis by blood spread, or into a central bronchiole to give us tuberculous pneumonia. In such case, the patient has had no previous infection, and so no immunity.

Blood spread generally results from repeated infections before the age of three. The patient gets no opportunity to acquire immunity, and therefore a resistance; he gets either one massive dose or continually repeated doses, usually from a parent with positive sputum. Probably there is no direct evidence of the lung involvement, which is inferred from the general condition, suspected by the history of contact, and generally easily confirmed by X-ray examination. The film shows multiple deposits of homogeneous, but ill-defined, small shadows like snowflakes throughout all zones of the lung fields; these are caseating tubercles (see Fig. 79).

The broncho-pneumonic type of spread infers some acquired immunity provoked by repeated small infections, but broken down by some intercurrent debilitating disease such as measles. That there is some resistance is indicated by the attempt to confine the spread, by inflammation and consequent fibrosis, in the peribronchial connective tissue, comparable to what we saw happen in the pathogenesis of streptococcal broncho-pneumonia as against pneumococcal lobar pneumonia. The film differs from that of simple uncomplicated broncho-pneumonia in two ways; first in its distribution, and second in its type of individual shadow. It is much more widespread, not mainly in the lower zones, and each shadow, while more woolly, is much more discretely marked off from its neighbour, especially on distant viewing, since the translucent spaces between are due to complementary emphysema in response to collapse of nearby affected lobules.

When immunity is swept aside by one massive super-infection,

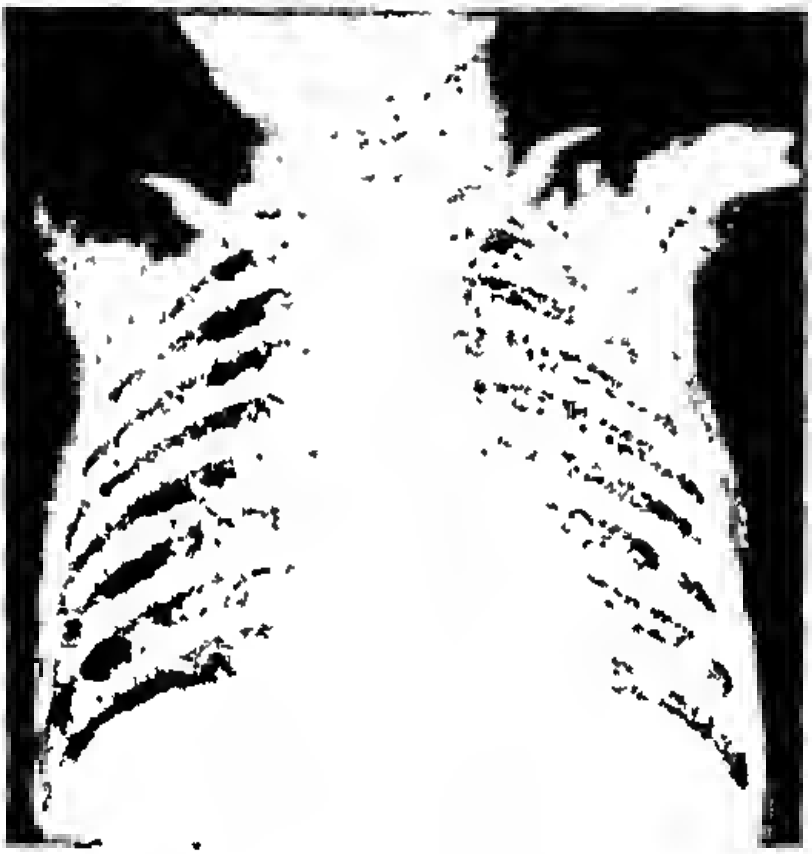

Fig. 79. Miliary tuberculosis.

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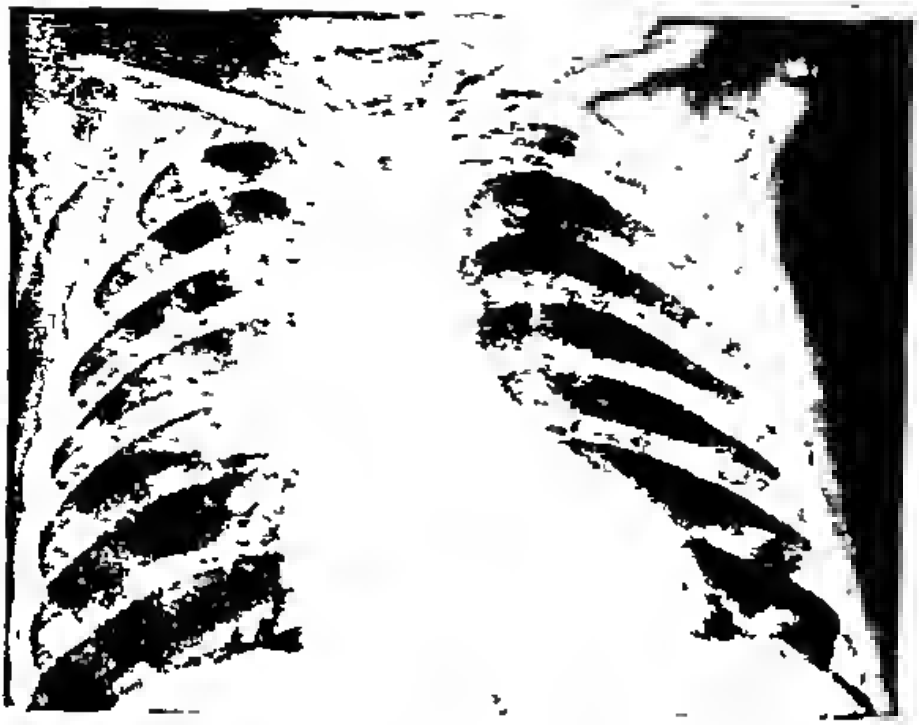

Fig 80. Epituberculosis. Acute stage showing hilar reaction merging with original primary focus.

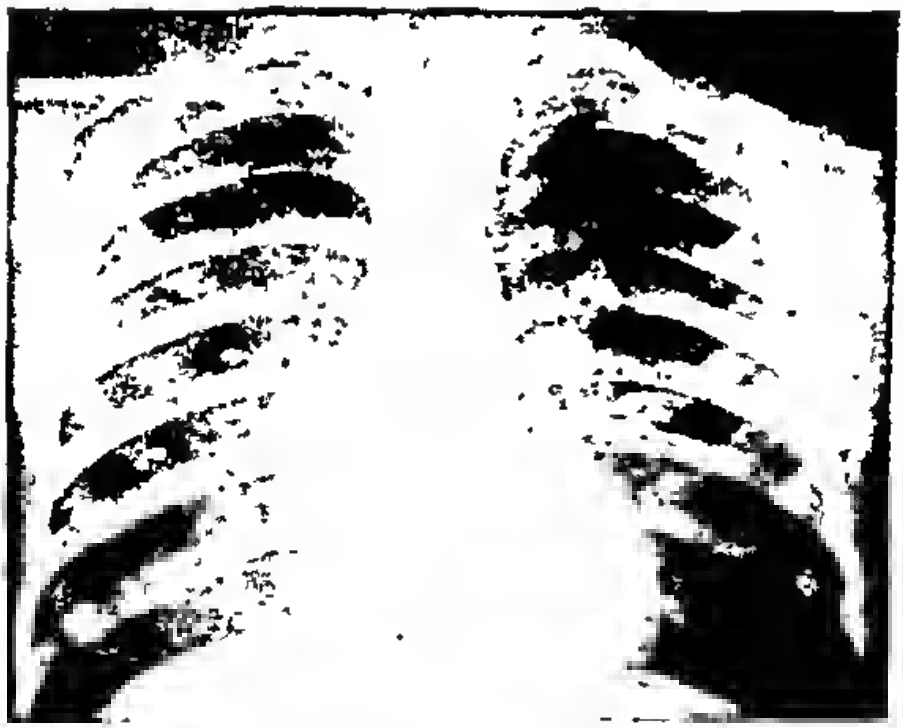

Fig 81. Epituberculosis. Same case as Fig. 80, four months later, showing healing with original Ghon's focus and consequent hilar glands.

then we have the condition comparable to lobar pneumonia in true tuberculous pneumonia. There is no evidence of fibrosis. This usually occurs under the age of five, and is for all practical purposes similar to the acute exudative phthisis of adolescence.

Tuberculosis in Later Childhood, up to Early Adolescence

In this country many children show acquired immunity by fibrosis, induced in response to earlier lesions. They show little, and generally no recognisable, clinical upset, and it is by chance that evidence of calcified deposits are discovered when a film of the chest is taken, usually during the investigation of some inter-current and unconnected illness. The pity is that these dead, and therefore clinically unimportant deposits are sometimes interpreted as the cause of the illness, with unfortunate repercussions to the patient and his parents.

Minimal re-infection at this period of life produces the first recognisable, so-called "primary focus." It is commonly known as the "Ghon's focus," showing up as a small, calcified, irregularly rounded area, usually well out in the lung periphery. It always drains, in the manner already described, to the associated glands in the hilum, which act as sumps for the region, and in many instances these glands are found to be enlarged and crenated by calcification. Absence of infection cannot be concluded from the absence of either a primary focus or enlarged glands on one postero-anterior film, *e.g.*, the focus may be hidden in the posterior recess by the diaphragm. When both are seen, with or without signs of inter-connecting streaks of irritated lymphatics, they are given the name of "Ranke's Complex." The finding of glands alone used to be read as evidence of the mythical "hilum tuberculosis," which is a misreading of the normal physiology of lymphatic drainage as well as of the pathogenesis of tuberculosis. This is, of course, an entirely different thing from the possible after-effects of those glands; the point is that they are themselves secondarily, and not primarily, infected. To such after-effects we shall pay some attention later.

We have seen how every clinical manifestation of the disease depends on the interplay of dosage and resistance induced by previous dosage. The infection can at this age group be so acute that there is complete breakdown of immunity, and so no fibrosis, in an unrestricted caseation. Again, the immunity may be lessened but not completely destroyed. so that we can find

forms that resemble the fibro-caseous type of adult life. Further, the local reaction in one confined area may be so intense that swelling of the bronchial mucous membrane by inflamed lymphatics causes occlusion of the lumen. Atelectasis of the broncho-pulmonary segment it supplies is the result. Or again, the glands in the hilum may have so much to do in coping with the drainage, and become so large, that they press on a main bronchus, and give lobar collapse; or may caseate through the bronchial wall and give broncho-pneumonic tuberculosis by inhalation. These possibilities, however, are all secondary and consequential conditions; they are not the condition described under the term "Epituberculosis." The name may be unfortunate, but it describes a true entity of childhood tuberculous infection (see Figs. 80 and 81).

Here there has been a superimposed infection of moderate intensity; the resistance has been maintained, while the actual amount of drainage has been more than the associated glands could deal with. It is just as if all the drains connected with the original focus, and all those normally emptying into the affected gland, were made to take the overflow, which therefore now begins to manifest itself from the hilum towards the periphery. The lymphatic drainage from one set of lymph channels is dammed back into every channel connected with the gland. The process therefore now simulates the radial spread of pneumococcal pneumonia, and may be misread as such. The first triangular loss of translucency round the gland is known as the "hilar flare." As in pneumonia, this spreading loss of translucency may go all the way to the periphery and even involve the pleura, but here any apparent similarity completely stops. Complete involvement and retrogression of the affected lung segment can take up to eight or more months, but we see that the method of resolution is exactly the opposite to that of pneumonia. The latter clears in the same order in which it extended so that it recovers at the hilum first and at the periphery last. In epituberculosis it clears at the periphery first and at the hilum last.

The temperature is subfebrile as a rule, unless pleural involvement supervenes. The child is fretful but of good general condition, and usually so little upset that he is considered to have a common cold or some teething trouble. Usually an X-ray examination produces the true diagnosis, although careful physical examination will show lack of movement, impairment

of note and weak breath sounds. There is nothing comparable to collapse, no shift of the mediastinum and so no sterno-mastoid sign. This is an acute lymphangitis, totally different from the possible secondary atelectasis of pressure or caseation involving a main bronchus. There is no reason why it should produce collapse in its usual uncomplicated state, for it is not a disease in the alveoli but in the lymphatics. The tubercle bacilli do not enter the vesicles although they irritate their walls and cause inflammatory oxudate, and, we have already noted, as this is only in its effects a substitute for the residual air, there is no mediastinal shift. A lateral film will confirm this.

The usual end-result is therefore a well-marked "Ranke's complex," and there is no proof that a child or adult so infected is any more liable than his colleagues to develop adult phthisis.

The film shows a generalised loss of translucency throughout the region of glandular drainage, entirely homogeneous in most cases, although occasionally a film of more than usual penetration may disclose the heavier shadow of the "Ghon's" focus towards the outer zone, in which it commonly lies. The upper zones of the lung fields are, by far, the commonest affected in the disease, and we can often see enlargement of glands that have picked up the infection across the mediastinum in the opposite hilum, though these will in many cases be easier to recognise on a lateral than on a postero-anterior film. The picture and the physical signs are entirely different if the complication of collapse has taken place. As we saw, this can occur in two ways. The localised lymphatic enlargement round a bronchus may be so intense that the broncho-pulmonary area supplied by the bronchus loses its residual air. We will now have a localised dense shadow on the film, and a drift of the mediastinum towards it. Again, the hilar gland may get so large that it presses on a main branch of the bronchus, and we shall have a picture of the same type as in adult lobar collapse.

A further complication is possible. This gland may caseate into the bronchus and the tuberculous material inhaled give a true broncho-pneumonic phthisis.

CHAPTER XVII

ADOLESCENT AND ADULT TUBERCULOSIS

"EPITUBERCULOUS" lesions are found up to about the fifteenth year of life, although most of them occur before the age of twelve. Thereafter we come to the manifestations of tuberculosis most commonly met with in general practice. Fundamentally they are the same as those of earlier ages in their interplay of infection and immunity; that is, they are probably due in the most part to reinfections or reawakenings of previous primary infections. It is highly possible, however, that many of them are continuing primary infections in individuals who have not been infected before the age of adolescence. Some patients show exudation with no foregoing, or with destroyed foregoing immunity; others show lessened immunity and thus varying amounts of consequent fibrosis. Some therefore show the spread of the blood-borne type of miliary tuberculosis; some show this form combined with fibrosis in the individual scattered lesions of "chronic miliary tuberculosis," while others show break into a bronchus and give broncho-pneumonic or pneumonic types. Most, however, show the disease commencing, as far as we know by their diagnosis through X-ray films, in those special broncho-pulmonary segments of dorsal branches of bronchi which we studied under applied anatomy, and saw involved in the commoner sites for formation of lung abscess. Considerable fibrosis is the usual response, so that fibro-caseous disease, and a tendency to consequent cavity formation, is the finding in the majority of patients. We do not know if such usual adult manifestations are really a secondary or tertiary stage of the disease, because we have no mass X-ray observations under the age of seventeen or eighteen. Similarly, we do not know what really happens to a primary infection in adolescence or adult life. Recent experience suggests that a young adult who is negative to tuberculin and gets first contact with an open case of tuberculosis shows film findings exactly like those which can accompany an erythema nodosum (see Fig. 82). There is a large confused hilar shadow, often with scattered indiscrete pulmonary shadows. These latter may be areas of lymphangitis and consequent collapse of bunches of alveoli. Some are complicated by pleural effusion. It is not

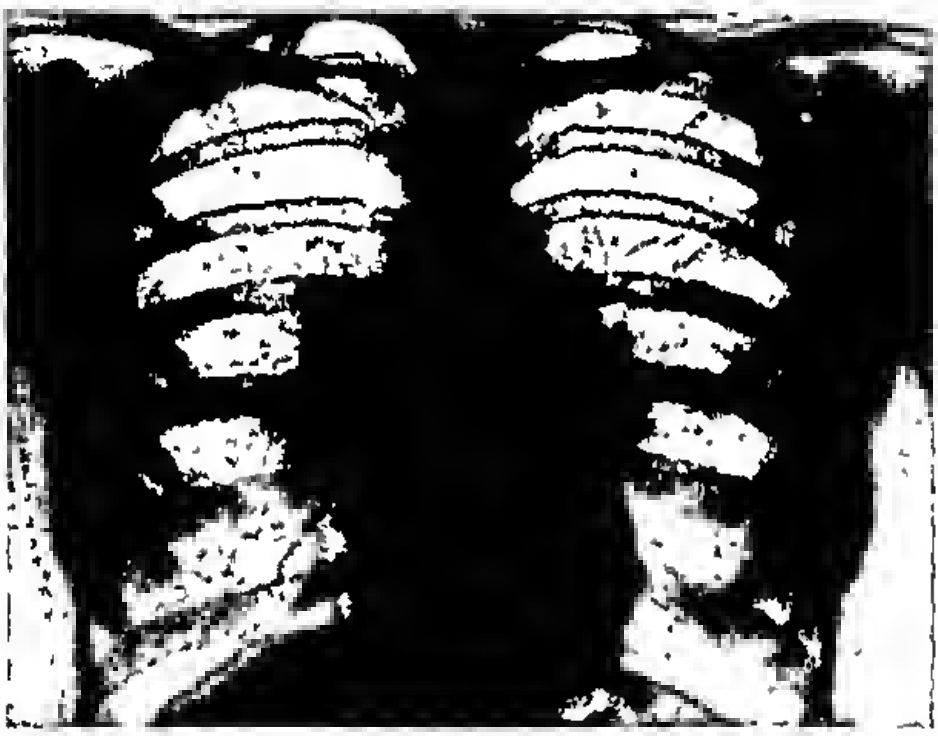

FIG. 82. The X-ray appearances which can accompany erythrema nodosum

FIG. 84. Tuberculosis in the axillary portion of the right upper lobe.

uncommon to recover tubercle bacilli from the stomach washings. The X-ray such as shown in Fig. 82 is usually read as "Sarcoidosis of lung." The writer has followed some such cases by serial films through a stage of fleeting shadows in one or both apices to definite infraclavicular deposits, with positive sputum, within eighteen months of the appearance of erythema nodosum.

In the involvement of broncho-pulmonary segments the attack is again through the lymphatic system; there is no change in the fundamental pathogenesis of the disease. Our studies on the anatomical relations of the alveoli reminded us that they connect with the lymphatics through lymph spaces, which surround the lobules. It is into these spaces that the phagocytes containing the tubercle bacilli now enter, after being caught in the lymphatics which accompany the dorsal bronchioles, and the first reaction is catarrhal exudate in the alveoli from irritation of their walls. This is a non-specific inflammatory œdema, the same reaction which we saw in epituberculosis.

It is well at this point to consider again, and sum up, what we have learnt about this exudate. It occurs in pneumonia and in broncho-pneumonia. We saw it also in the lobules adjacent to those filled with the septic material producing lung abscess; now we find it in the first reaction of the alveoli to the irritation of the tubercle bacilli in their surrounding lymph-spaces. It always gives a homogeneous shadow, and is similar to that produced by the congestion of "congestive failure," which is exaggerated in œdema of the lung. It is the confused edge of all film shadows of inflammatory chest conditions, and can often obscure the underlying lesion, *e.g.*, an early tuberculous disease. It has been given the name of "pneumonitis," which name can do no damage provided it is understood that it is not a diagnostic term but only a descriptive name for a non-specific œdema, the true cause of which must be sought by further investigations.

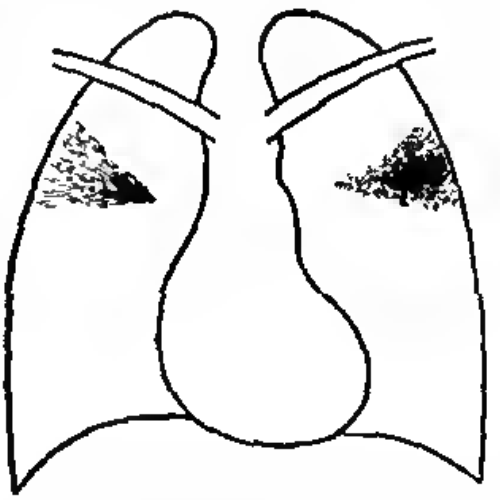

Fig. 83. Tuberculosis left outer mid zone, compared with pneumonia of axillary branch of the right upper lobe bronchus.

It is of particular importance that its shadow in adult tubercle of the outer mid zone should be recognised; in this position it often covers for a considerable time many small individual shadows of infected acini. We have already compared it for differential diagnosis with pneumonia of the axillary branch of the upper lobe bronchus (Figs. 83 and 84).

As this non-specific œdema replaces the residual air of the alveoli which it fills, it has the same physiological effect on the mediastinum; in other words, it is never in itself the cause of mediastinal displacement. It follows, therefore, that in the earliest tuberculous reaction there will be no sterno-mastoid sign and no added sounds, while there will be lack of movement.

At this stage of the disease, and indeed for weeks or months, even in a progressive lesion, the patient may make no complaint, and should he be examined, can present no definite abnormality. Only X-ray examination will reveal the disease, and only serial examinations by film may be the deciding factor on an ultimate decision of activity or inactivity in any one individual case.

We learnt under applied anatomy that the smallest branch of the bronchi is the bronchiolus respiratorius; each supplies an acinus. As each acinus is surrounded by a lymph channel, it is this anatomical division of the lung parenchyma which is fundamental to our understanding of the further changes in adult tuberculosis. Each goes through all the stages, in its component alveoli, of catarrhal exudate, then invasion of this exudate by the tubercle bacilli, then caseation, and then the attempt to evacuate this caseation into the bronchiolus. Blockage of the bronchiolus follows, so that those alveoli that have emptied collapse by loss of residual air, and those that cannot now empty organise. Next will inevitably come dragging on the wall of the bronchiolus, already weakened by the tuberculous material, so that cavitation commences. This process will be aided by the increased pull of the intra-pleural negative pressure, seeing the elasticity of the infected acini is now being destroyed, and at this stage we find the sign of mediastinal displacement in a tense sterno-mastoid muscle on the side of the lesion.

We may now follow these changes on the X-ray film. The dorsal bifurcations of the pectoral and axillary branches of the right epi-arterial bronchus are commonly affected. This is why it is not uncommon to find the first recognisable foci of adult tubercle as rounded areas of homogeneous loss of translucency in

the inner or outer thirds of the right infra-clavicular region. They are round because, as we have noted before, the cone of infected parenchyma is photographed through its long axis. They may be in small and multiple areas up to about ½ inch in size when first noted, but are usually single and about three times this size. They are then known as "Assmann's foci." They are fairly well defined, and entirely homogeneous. The apparent striations on them are really the normal markings of lung tissue lying anterior to them on the postero-anterior film, as can be shown by taking either an antero-posterior film which presents them nearer to the surface, and therefore to the film, or by doing a tomograph (see Fig. 43, p. 61).

There is one reaction to the focus that is quite common in the upper lobe, especially the right, and is often called erroneously "tuberculous pneumonia." We saw that in "Epituberculosis" there is a possibility that lymphatics round a bronchus can be so swollen by reaction that they occlude the lumen, and produce a localised area of collapse as a complication. A comparable reaction can take place in response to an acute "Assmann's focus." Swelling in the lymph-spaces, together with much catarrhal exudate in irritated alveoli round them, can give a heavy shadow, part of which is due to actual lobular collapse. The interlobar fissure is drawn up and the mediastinum well displaced to the affected side. The lymphatic inflammation is quickly passed to the interlobar fissure, which is always heaviest in its shadow just below the lesion. This is the condition that was compared for differential diagnosis with upper lobe atelectasis (see Fig. 61, p. 85)

Rest and serial films will often show the true state of affairs. Just as the catarrhal reaction often disappears round foci in other parts of the parenchyma, now both the catarrh and the lymphangitis can be seen on serial films to retrogress to more and more translucency, while in the rounded area of the causative deposit we not infrequently see a rapidly forming cavity with a fluid level. The condition has been, therefore, but a manifestation of the severity of the attack.

The temperature and constitutional disturbance may simulate pneumonia at onset, but the dullness of the upper lobe is more posterior than anterior, and we shall find signs of mediastinal displacement, which, as we have seen, can occur only in complications of pneumococcal pneumonia. Later, the film can

simulate abscess formation in a lobe collapsed by bronchial carcinoma, but as the cavitation develops in the tuberculous case the rest of the lobe clears above the lesser fissure, whereas, in true carcinomatous atelectasis, the surrounding density remains. By this time sputum examinations will generally clinch the diagnosis.

The large rounded form of the Assmann's focus is, we have seen, by no means a necessity. Individual acini may be seen on the film as tiny individual dot-like foci congregated together, and giving no evidence of inflammatory exudate. We have already noted that such single or multiple foci can be concealed by overlapping bony shadows. Where there is any doubt the rib intersections must be studied very closely and an antero-posterior view taken. All this means is that there is less acute invasion, or that we are looking at a later stage when the exudate has gone; it does not mean that these areas will not go on to the same after-effects. Serial films show they can proceed to cavitation of slower onset, although they are, on the whole, more inclined to go on to organisation and fibrosis without much drag on the bronchus. If, however, they caseate and affect by extension other acini in their immediate neighbourhood they are no different in their film findings from those Assmann's foci which appear to break up into their constituent parts and produce a picture of small triangular areas that merge into the shadows of the inflamed lymphatics, round congested bronchi tracking towards the hilum. The disease is following its constant pathogenesis; it is attempting to drain by the lymphatics to the hilar glands.

This type of individual acinar deposit is common in the supraclavicular apex, and means that the posterior apical branch of the upper lobe bronchus has been affected (see Fig. 13, p. 7). Experience with mass radiography shows this is a far commoner site of disease than was formerly believed, and many cases that show active Assmann's foci below the collar bone have evidence of previous true apical deposits. Indeed this is so common that it appears probable that infra-clavicular tubercle is a second or perhaps a secondary attack to a previous and unsuspected more peripheral disease, never quite healed. Whether such true apical tubercle is an outpost of the primary focus of childhood that remains dormant until adolescence we may, by the modern possibilities of serial film examination, soon be able to state.

It can be extremely difficult to decide whether such deposits

are present in the supra-clavicular apex. Overlapping bony shadows, the shadow of the sterno-mastoid, pleural tenting and pleural striæ can all give confusing shadows. The antero-posterior view is often of great help in the final opinion (see Fig. 28, p. 31).

If we now looked at the cut surface of the post-mortem specimen we should see a collection of tiny nodules of collapsed and organised acini, each round a central, more or less occluded, bronchiole. We might also recognise many white strands due to fibrosis of the peribronchial and perivascular connective tissue, the latter narrowing the blood vessels and helping to contribute to the endarteritis obliterans which is a constant feature of the disease. The hilar glands are hardly, if at all, affected; the fibrosis has allowed but few bacilli to reach them.

It is little wonder that tuberculosis is without symptoms or gross physical signs, especially to stethoscope, for a considerable time after its establishment in adult form. It does produce lack of movement, however, and, very soon afterwards, destruction of elastic tissue begins by organisation and the results of bronchial blockage. The sterno-mastoid sign reflects drag on the mediastinum, but only careful stethoscopic listening will demonstrate the first sound of the material evacuating from the lobules into their supplying bronchioles. We shall then hear evidence of the bronchiole contents in fine sibili at the very end of inspiration, increasing in intensity to reach a crescendo at the very end. Remembering our stethoscope findings in bronchitis we recognise a distinctive and diagnostic difference in the position in inspiration of this sound, which is exactly the same in its quality to the ear in both diseases. In bronchitis we heard it at the beginning of inspiration, lessening with the increasing depth of breathing, and connoting obstructive material consisting of breaking down elements of the wall; what we hear in phthisis is material evacuating into the terminal, distal ends of the bronchioles from their connecting lobules. With these sibili or coarser rhonchi we shall soon hear the dry crepitations of fibrosis, also most marked at the end of inspiration, and not disappearing with cough; these are the signs of the peribronchial fibrosis. In other words, we have stethoscopic evidence of infiltration and fibrosis (see Fig. 85).

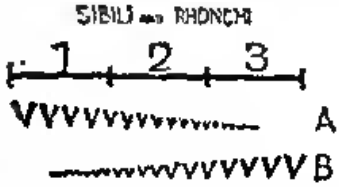

Fig. 85.

A. Bronchitis
B. Pulmonary tuberculosis

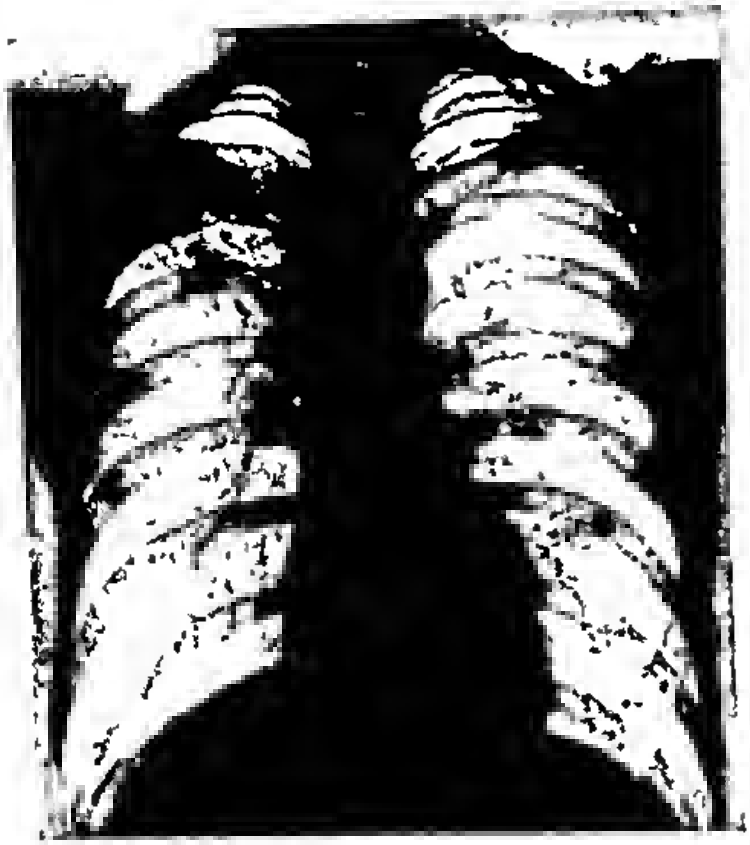

Fig. 87. Tuberculous infiltration and fibrosis. P-A view.

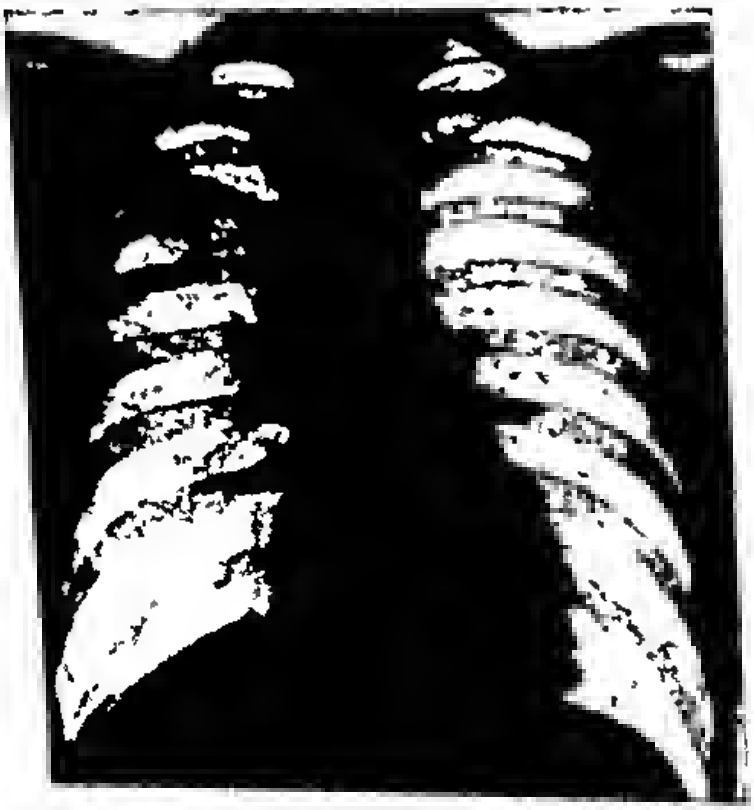

Fig. 88. Same case as in Fig. 87. A-P view shows a cavity in upper [illegible]

Thickening of the pleura overlying the broncho-pulmonary segment follows very soon. Its stethoscopic sign we shall also find, in a burst of fine dry crepitations with the act of cough which disturbs the fine adhesions of organising exudate between the visceral and parietal layers (see Fig. 86).

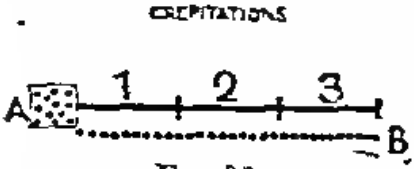

FIG. 86.

A. With act of cough in pleural thickening.
B. With fibrosis of lung.

The progress of a focus that keeps its rounded form for some time shows by serial films that it may, although rarely, seem to disappear altogether. It may split, as already noted, into small individual lesions, or go on to a drawn-out mass of fibrotic strands and organised areas. Sometimes there will be rapid cavitation, with occasionally a fluid level, at first X-ray recognition. Usually, however, the cavity is due to a slow weakening of the walls, which are dragged peripherally in ragged fashion by organisation here and collapse there, so that the film shows an area that has lost in great part its rounded formation, and is merging into the shadows of the larger bronchi, now outlined by surrounding thickening connective tissue, as they track downwards and inwards to the hilum.

Critical inspection of the film of an established case with activity gives us a lesson in living pathology. If we look at it closely we see it has a background of fine generalised loss of translucency; this is thickened plastic pleurisy. At the edges of the lesion we see areas of slightly greater loss of translucency; they are due to the catarrhal exudate in irritated lobules in the process of invasion, *i.e.*, the areas of advancing infiltration, merging into the heavier shadows of lobules now containing tubercle bacilli, that have penetrated their walls, and are producing caseation. More centrally we see still denser individual lesions, clear cut, standing out on distant viewing like the thickened bronchial walls, because they have round them lobules distended by complementary emphysema. These denser areas of collapsed and organised lobules are fused and solid in clumps, round the dilated and destroyed bronchial wall, to form the heavy, but broken up, outline of cavity. The picture is distinctive and diagnostic, unlike any other in chest disease (see Figs. 87 and 88).

There is possible confusion with the cavity of true lung abscess, but, as we have already seen, the cavity surround in this case looks

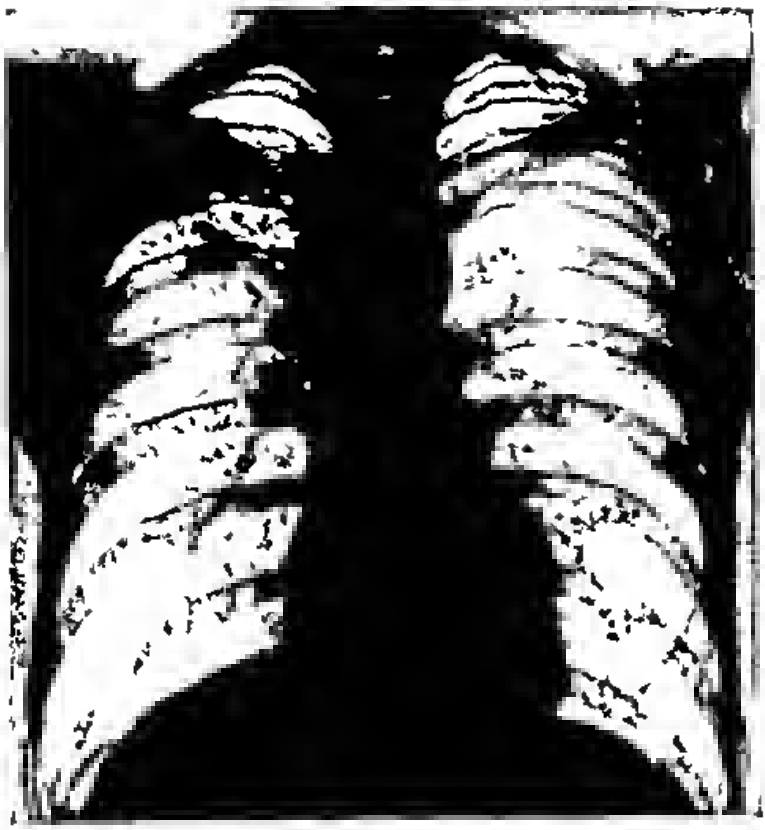

FIG. 87. Tuberculous infiltration and fibrosis. P-A view.

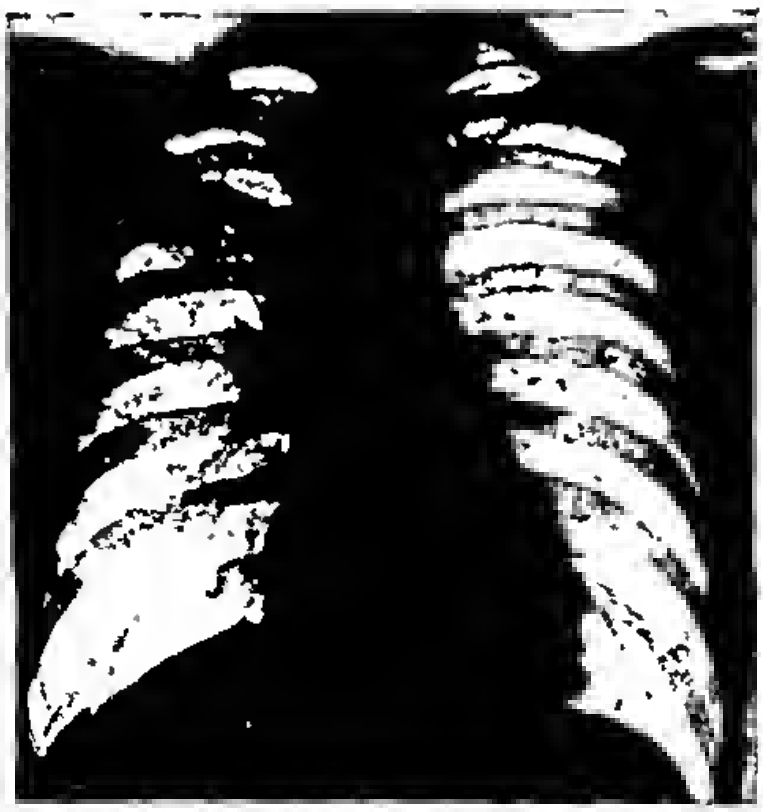

FIG. 88. Same case as in Fig. 87. A-P view shows a cavity is present.

[To face p. 110.

FIG. 90. Tuberculous cavitation.

as if all the affected lobules had been suddenly frozen *in situ* to form a continuous band with no areas of intervening lobular emphysema, and therefore not altering essentially in their detail on distant as against close viewing. We saw also that a further and important point in such differential diagnosis is that the chronic tuberculous cavity is seldom the only evidence of phthisis; it is much more often in a lobe throughout which can be seen scattered areas of infiltration with fibrosis (see Figs. 89 and 90.

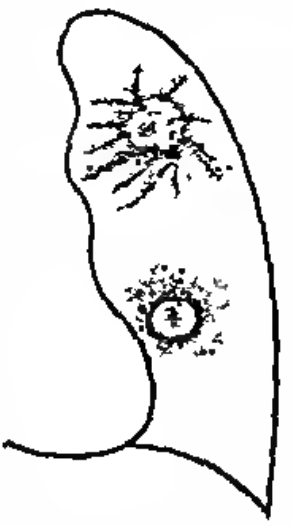

FIG. 89. Cavitation in pulmonary tuberculosis.

The advent of the cavity gives its own adventitious sounds. The presence or absence of whispering pectoriloquy is not a reliable diagnostic finding; the more the contents of the cavity at the time of listening, the less the pectoriloquy. A much more constant sign is the presence of coarse, metallic râles (see Fig. 91). They are in quality exactly as those we heard in bronchiectasis and in lung abscess, but they are now heard beginning in the second half of inspiration, and become more and more insistent with the depth of breathing, until they reach a crescendo at the very end.

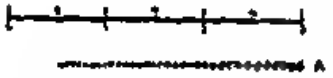

FIG. 91. Metallic râles in tuberculous cavitation.

We must remind ourselves that considerable portions of lung tissue are involved before added sounds are heard. It is a fairly safe rule to conclude that signs heard above the clavicle mean the upper third of the lung is involved, and signs below the clavicle mean at least half of the lung is involved. An example of the influence of other factors in their production is that where laryngeal tubercle is present we can consider the extent three times that indicated by our stethoscope findings.

Acute Exudative Phthisis of Adolescence and Adult Life

This type of pulmonary tuberculosis is fortunately nowadays rare in this country, as acquired immunity is widespread. We do, however, occasionally see it as a steadily advancing disease unrestrained by any fibrosis. Inflammatory exudate in the lobules is rapidly replaced by caseating material, which on evacuation

fills the bronchi, whole areas breaking down to be coughed up and to leave in the lung fields ragged vacancies, which are therefore cavities of entirely different mode of formation from those we have already considered. As no barrier is placed in the way of lymphatic drainage, tubercle bacilli flood the hilar glands, and they in turn go on to rapid caseation.

The film therefore shows many areas of translucency among widespread homogeneous densities. The translucencies are the cavities, often of no clear definition, but occasionally looking like ticket punchholes where there has been a sudden evacuation, through a bronchiole, of rapidly caseated tuberculous material round it. Bunches of heavily infiltrated lobules then make up what is only an apparently confining wall. Hilar shadows are heavy, indistinct and confused, made up of the caseating glands, surrounded by much swelling in lymphatics, and much increased hyperæmia.

Movement of the chest is poor; there is generalised loss of note with no offsetting areas of emphysema. The predominant part of the affected zones of the lungs is silent, as residual air is replaced by exudation and caseation, but now and again we hear coarse rhonchi, caused by the bronchial obstruction. These are especially noted, being exaggerated and insistent even after cough, in areas going on to cavity formation by rapid evacuation. We are listening to "tuberculous bronchitis" which is really a flooding of the bronchi by the evacuating caseated, tuberculous deposits.

CHAPTER XVIII

PLEURISY

THERE are three main forms of pleurisy: "fibrinous," or dry; "sero-fibrinous," or pleurisy with effusion; and "purulent," or empyema. There is no hard and fast delimination between these forms, seeing that there is always some slight fluid present with the first, while the second and third really vary only in the amount of pus in their cellular content. All three forms can be caused by the pneumococcus, the streptococcus or the tubercle bacillus.

Dry pleurisy may be primary to the pleura by the presence in its layers of the bacillus of Koch. It is however almost always secondary to pulmonary tuberculosis if it be tuberculous, even if it is actually found in the parietal layer, in which case what has happened is that bacilli from a previous frank effusion have penetrated this layer to its intimal coat. Dry pleurisy is also found with pneumonia and broncho-pneumonia and their suppurative complications, and may be diaphragmatic in site when secondary to abdominal disease, which is commonly subphrenic abscess.

Its effect on the membranes depends on its cellular content, the higher the polymorph percentage-count, the less being the chances of ultimate and permanent damage. Thus is why lobar pneumonia, while producing a heavy exudate on the visceral layer, yet leaves little ultimate damage in the vast majority of cases, as its greyish-yellow, thick deposit, even should it undergo some organisation, will be removed eventually by the proteolytic enzyme of its preponderant polymorph content. On the other hand, tuberculosis produces an exudate which contains a high percentage of fibrin as against polymorphs, and so is much more liable to proceed to permanent adhesion between layers of the pleura over parts of the lung affected by phthisis.

We can follow these effects of dry pleurisy on the film. We see much more density from a recent pneumonic than from a recent secondary tuberculous pleurisy; but the pneumonic one, followed in serial films, will almost always disappear, while the tuberculous one leaves behind a haziness of fine homogeneous type, to act as a background to those lung shadows of infiltration and fibrosis which are the underlying cause of its appearance.

There are two exceptions to this rule, the first being demonstrated in so-called "tenting of the diaphragm." We saw already that localised lung affections of pneumonic type are slow of disappearance, and incline to healing by repair rather than by resolution as against their more generalised forms. The same effect is seen in interlobar pleural infections. We have noted earlier that very often a localised tent-shaped shadow is seen on a film about the mid-point of the diaphragm on one side, more often the right than the left.

We noted the second when discussing the diaphragm. It consists in that filling in of the extreme costo-phrenic angle by a shadow which has no clinical significance unless it be accompanied by a lung focus. Without evidence of such a focus it should never be read as tuberculous in origin. It is almost always due only to a previous known or unrecognised pleural reaction of pneumonic or broncho-pneumonic origin.

The most painful type of dry pleurisy is the pneumonic. The progress of the average secondary pleurisy, whatever its cause, is from visceral to parietal layer, each being involved first by inflammatory reaction and then by the specific reaction of the penetration of the causative organism, in the same way as we saw the progression of any lung disease involving the alveoli, from catarrhal to specific exudation. The pneumococcus first irritates, and then invades the visceral pleura as it reaches the periphery of the lung sector; it seldom does more than inflame the parietal layer. Œdema stretches and makes this layer supersensitive. Dragging by adhesion between the moving visceral and the static parietal layer then produces great pain. With lung abscess pain is not frequent, and with plastic pleurisy of tuberculous origin the patient complains more of a "rheumatic ache" than an acute discomfort, unless as in pneumonia he has a widespread reaction to the parietal layer, in which case he is almost always about to develop a frank effusion.

As already noted, sero-fibrinous effusion differs from dry pleurisy only by virtue of the fact that fluid is in amount sufficient for physical or visual demonstration. The cause in most cases seen in general practice is tuberculosis of the lung. It is to this latter type that we shall confine our detailed description of frank pleurisy.

The fluid contains much fibrin, a high percentage of protein, and a preponderance of lymphocytes as against polymorphs,

when compared with the high polymorph content of the pneumococcal and the rarer streptococcal effusions. It is usually clear, and has an opalescence due to suspended fine particles of fibrin, easily seen in fluid examined *in situ* through a thoracoscope, and quickly forming the typical clot in samples withdrawn for visual and laboratory examination. It is not cloudy like the pneumonic exudate with its polymorphs tending to form pus. In the fibrinous content lies the dangerous liability to the formation of thick coverings on the visceral and parietal layers, when the fluid is left to organise in the pleural cavity. Tubercle bacilli seldom appear in smears and may fail to appear in culture if the sample is examined under four days from the onset of the effusion; invasion has not yet followed irritation and consequent inflammatory exudation. Failure to realise and act on this simple fact of pathogenesis may have serious consequences for the patient, by inefficient treatment founded on faith in one laboratory examination report which has been made too soon, and without clinical notes to the pathologist.

To physical examination there will be dullness beginning in the lower axilla and extending upwards with the increase in effusion. While there will be silence in the area displacing the lung, there will naturally be increased breath sounds over that part of the lung, on the limits of the fluid, which are by it pressed inwards and upwards; the collapsed lung acts as a sounding board. The outstanding point is, however, the displacement of the mediastinum to the opposite side, demonstrated in a positive sterno-mastoid on that side, and usually supported by evident displacement of the apex beat to palpation and auscultation.

We may remind ourselves how the lung reacts to the presence of fluid in the pleural cavity. When the negative intra-pleural pressure is brought nearer to that of the atmosphere there will be less outward pull on the underlying lung. Its elasticity will come into play, therefore, and draw it inwards to the anchorage of all elastic tissue at the hilum. From the first the reduction of the pleural pressure on the affected side will make that on the opposite side greater in comparison, and so the mediastinum will be first of all drawn to this opposite side. With more fluid the pressure in the pleural cavity will be greater than that of the atmosphere, and so begin to bear in on the parenchyma; that is, it is greater than the pressure of the residual air in the lobules. But we can see that there does not need to be any actual push from the

affected side in order to give a positive sterno-mastoid on the other.

After this point the reaction of the lung and the mediastinum depends on whether they are healthy or already diseased. A lung with preceding fibrosis will naturally resist collapse, and will move bodily against the mediastinum, while a fixed mediastinum, *e.g.*, one that has been affected by previous pleurisy, will resist displacement. What this means is that we must consider all these separate factors, and not conclude that the amount of mediastinal displacement is in anything like exact ratio to the amount of fluid or the actual change in pressure in the intra-pleural space.

While it must be stressed that no pleural effusion has been fully investigated unless exploratory puncture for visual and laboratory diagnosis, and blood film examination, have been carried out, it is none the less true that a tuberculous exudate has a characteristic film finding (see Figs. 92 and 93). In its early days it has a specific fine loss of translucency through which the ribs can be seen almost as clearly as they would be with the presence of a pneumothorax. Towards the mid zone from above downwards there is a comparative density concave inwards, running from the axilla to the diaphragm, but this is not in the uncomplicated case a clear, single line of demarcation. It is made by the combined shadow of fluid and lung, the former lying to some depth laterally both in front and behind the latter. If there is no underlying lung disease we ought to be able to see exaggeration of the normal vessel markings to the inner side of this junction, owing to the hyperæmia in the lung contracted towards the hilum. If these markings cannot be seen we must suspect consolidation from, for example, pneumonia (see Fig. 94).

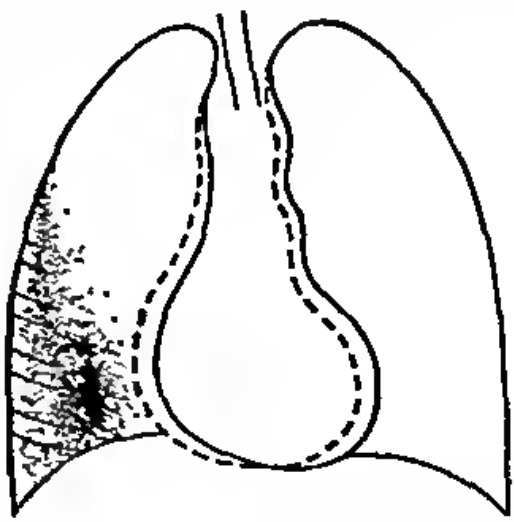

FIG. 92. Tuberculous pleural effusion.

The picture is therefore not like that of uncomplicated lobar pneumonia of the lower lobe, with its density at the base, decreasing from the diaphragm upwards towards the lingula, and equal

FIG. 93. **Tuberculous pleural effusion.**

[*To face p* 116

at any one level between the axilla and the mediastinum. In pleurisy there is increasing loss of translucency from the axilla inwards to the junction of fluid and lung. Moreover, it will be differentiated also by the position of the heart and the mediastinum, for we saw in our study of the pneumonias that they do not cause displacement.

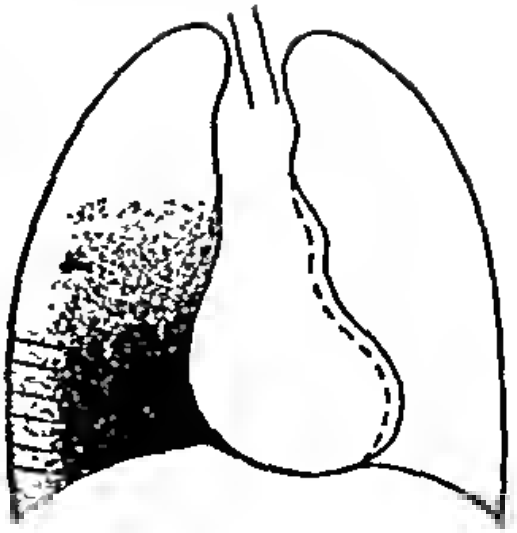

FIG. 94. Pneumonia right lower lobe, with fluid. Note loss of vascular markings, as in uncomplicated pneumonia, but displacement of mediastinum due to complicating effusion.

If fluid has been present for some time before the film has been taken, the concave density may have assumed a sharp outline. This is because organisation of the fibrinous exudate on the visceral pleura has already taken place, and the patient is thereby in danger of incomplete re-expansion of the lung, even with immediate and complete withdrawal of fluid. Should the effusion be of still longer standing, organisation of the fibrin floccules gives a dense white shadow occupying the area between the axilla and the now sharply defined border of the lung, fixed and compressed.

The shadow is not now anywise different from that of true empyema, although the latter in its acute stage will cause displacement to the opposite side. This will be a definite aid in visual diagnosis, but it must call for the accessory aids of blood and exploratory-puncture examination. The usual cause in general practice cases is the pneumococcus, for true tuberculous empyema is a rarity, and seldom met with except as a complication of a previous clear effusion, especially if such effusion has occurred as a hydro-pneumothorax.

The average case of such idiopathic pleurisy as just described clears completely with no remaining evidence in pleura or lung. If we see on the film some filling of the costo-phrenic angle and no lung focus is evident on a postero-anterior film, we ought to take lateral and lordotic films, and even tomographs, if we wish to be as certain as is humanly possible of our final opinion. Often the small tuberculous deposit is in the periphery in the upper

mid zone, or in the supra-clavicular apex from involvement of the posterior apical bronchus.

Interlobar effusions apart from that in the lesser fissure on the right side are seldom evident or demonstrated to their true extent in the postero-anterior film. A lateral shows them clearly, forcing apart the interlobar septum, and usually bow-shaped, because they are pressing on the parenchyma of both lobes (see Fig. 66, p. 89). Fluid in the main fissure can give on the postero-anterior film a shadow very like that of true diaphragmatic effusion or empyema secondary to abdominal disease, *e.g.*, subphrenic abscess. Both sit over the diaphragm and are inclined to follow its outline. The physical signs, however, will help us to differentiate, because while the latter gives us dullness both anteriorly and posteriorly, the dullness due to interlobar effusion is especially marked in front. A lordotic picture aids us too, for the true interlobar effusion rises through the middle of the lung field like a drawn-out tent sitting on the diaphragm.

The usual dry diaphragmatic pleurisy shows no evidence to film examination to account for the acute shoulder and upper arm pain that accompanies it.

Pneumothorax

From what has been said of the effects of pleural effusion on lung elasticity and mediastinal shift we can visualise the changes in pneumothorax without further detailing them. The effect on the film findings of the underlying lung is the same, and the diagnosis must rest on the alterations noted both within and without the lung field. As against pleural effusion we now have complete absence of shadow between the rib interspaces outside the line of the retracted lung; no loss of translucency and no lung markings are evident (see Fig. 11, p. 6).

In the "simple" or non-tuberculous case it is extremely rare for fluid to appear in the pleural space, and it may be possible to see on the edge of the collapsed lung emphysematous bullæ, one of which has burst to produce the pneumothorax. In the case where pneumothorax complicates an already demonstrable or undemonstrable lung tuberculosis, fluid is exceedingly common, and is always in its upper limit along a horizontal line in the erect position.

Tympany occurs in both conditions, and shifting dullness in the lower axilla accompanies it in cases of hydro-pneumothorax. Adhesions to the parietal pleura can produce the so-called "bell-sound" when they are under tension, whether the pneumothorax be spontaneous or induced.

CHAPTER XIX

CARCINOMA AND SILICOSIS

Carcinoma of the Lung

Carcinoma of the lung is common as compared with sarcoma, and although it is more usually a secondary than a primary occurrence, it is the primary form, probably always bronchial in origin, to which we shall make short reference here. We need not consider the types of causative cell found, but note only that they usually start with a mere interference with the bronchial mucosa. Later the lung lobules are slowly replaced by tumour spaces containing a mucinous exudate, or are irritated and thickened in their individual alveolar walls by the actual tumour cells.

Under the heading of atelectasis we have already made a detailed study of the effects of carcinoma of the bronchus on the affected lobe and on the postero-anterior film. A short note is necessary on the so-called nodular form because of the difficulties in differential diagnosis. For details the student must consult specialist treatises: all that is attempted here is to give some broad outlines for guidance. On the whole, clinical history and investigations alone can give the answer in a large majority of cases; X-ray is but an accessory aid.

Three main forms of nodular carcinoma may be mentioned: the mediastinal, the medial and the basal.

The mediastinal or hilar form originates near the division of the trachea into its main right and left branches. The growth invades the glands and all the mediastinal tissues to produce on the film a large, rounded, extremely opaque and homogeneous shadow.

It may therefore simulate the true glandular diseases: lymphadenoma, lympho-sarcoma and leukæmia. Of these the commonest by far is the first, which shows mediastinal glandular enlargement in about one-third of its cases (see Figs. 95 and 96). It may be noted first on the film on the right side, as if it were a swelling connected with the superior vena cava, but it is nearly always bilateral from its first recognition. Two points may

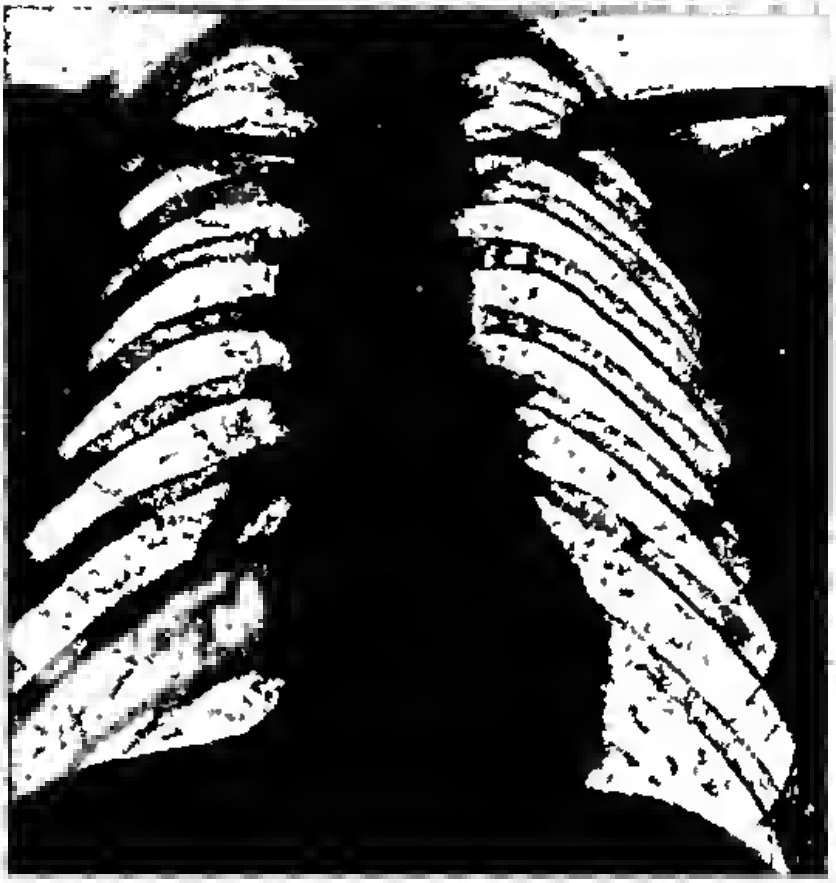

FIG. 95 Lymphadenoma

FIG. 96. Lymphadenoma. Oblique view of case in Fig. 95.

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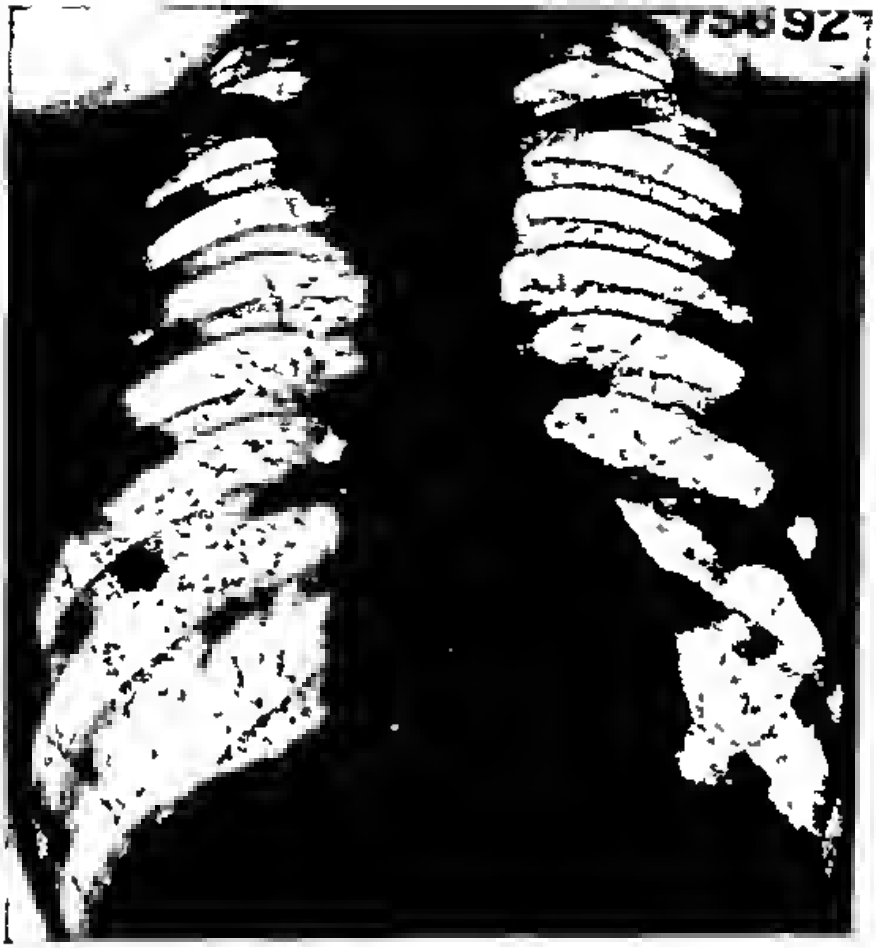

Fig. 97. Neurofibroma.

Fig. 98. Dermoid of lung.

aid: first, it is generally lighter than cancer in its loss of translucency; second, it is usually an upper mediastinal disease, and so definitely higher on the film than "mediastinal" cancer. Only later does it get ragged in its outline, as the "chronic" type begins to invade the parenchyma along the bronchi. At this stage it may be impossible by X-rays to differentiate chronic lymphadenoma and hilar carcinoma. Blood examination, search for other glandular involvement, and biopsy, may be necessary for ultimate diagnosis.

The same criteria must apply in diagnosis against lymphosarcoma and leukæmia. The response to radium and deep X-ray therapy on serial films is not a safe guide; all that can be said with certainty is that carcinoma is more resistant than the others to such test treatment.

These conditions are unlikely to be confused with the X-ray findings in mediastinal dermoid cyst and neuro-fibroma. The former is usually a clearly outlined rounded or oval shadow: most cases show by lateral film that the growth lies in the anterior mediastinum. Neuro-fibroma, which is the commonest benign tumour of the chest, lies in the upper posterior mediastinum (see Fig. 97). It too is usually well-defined and rounded in appearance. It arises from one of the intercostal nerves and throws a shadow only slightly more dense, on the average, than that given by the superior vena cava.

Only immunological and precipitin tests, including the Casoni test, may help towards final opinion as between neuro-fibroma, hydatid and dermoid cyst (see Fig. 98). Again, it may need thoracotomy to decide between neuro-fibroma and that type of nodular carcinoma which starts in one of the smaller bronchi and invades the parenchyma through the bronchial wall. This latter condition produces on the film an individual rounded opaque shadow, which it may be impossible to name even after full radiological examination.

The medial form is due to carcinoma of a main bronchus close to its origin. The shadow usually looks as if it were part of the main cardiac shadow, resting like a hump on the ventricular border. The outline may be sharp or quite ragged; it changes but little over a period of time to serial film examinations (see Fig. 99).

The basal form has an ill-defined lung shadow, attached to the hilum by many irregular streaks. It is usually of changing densities, as it has within it areas of obstruction of bronchioles,

and it is not uncommon to find it later go on to the usual picture of lower lobe complete atelectasis.

Silicosis (see Fig. 100)

There has been much recent interest in this disease as it is one of the commonest industrial affections of the lung. It is an example of the progress along the lymphatics of dust inhaled directly into the alveoli. In the alveoli it produces catarrhal cells which are phagocytic to it; these pass to the lymphatics of the lobules. Some stick there, but many are drained to the lymphatics of the lungs, there to be held in tiny clumps which grow by steady addition to them, and by the cellular reaction they produce. Fibrosis is the result, so that we can see on the film many scattered hard-looking nodules.

The stages to such nodulation can be followed on the film. First the irritation of the peribronchial and perivascular lymphatics gives us increased reticulation. Next, with blockage in lymph spaces towards the hilum, and at bronchial bifurcations, we can see tiny nodules appearing, clear cut by their surrounding complementary emphysematous lobules. Still later we find these nodules fusing into fairly large areas of consolidation, mainly round those same dorsal branches of the bronchi which we have seen affected in lung abscess and fibro-caseous tuberculosis of adult type. With these we find a characteristic fibrosis round the hilar shadows and extensive, gross emphysema throughout the lung fields.

Fig 99 Carcinoma of the lung—nodular (medial) form.

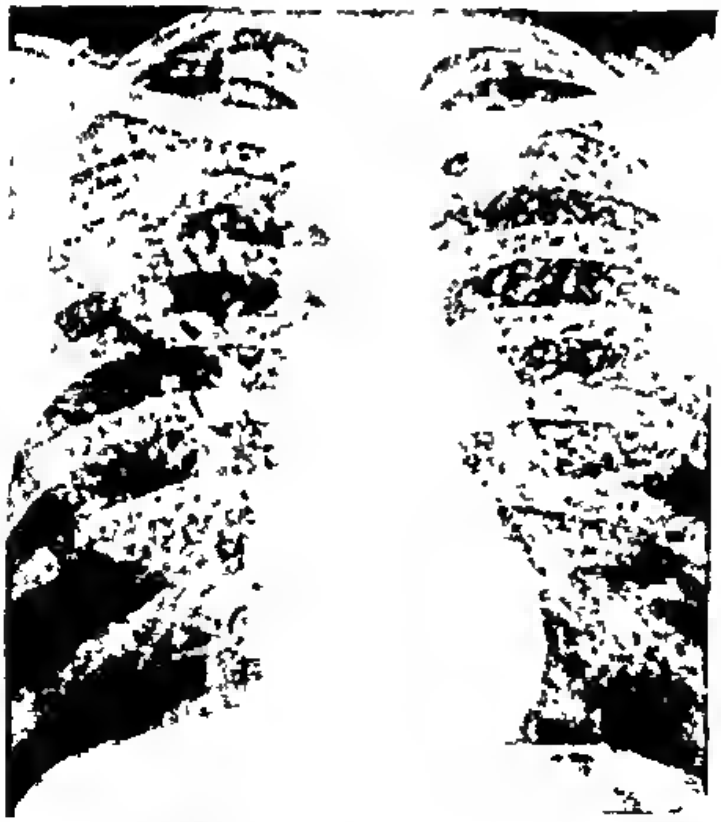

Fig. 100. Silicosis in a hæmatite miner.

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